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THE DIAGNOSIS AND TREATMENT OF THROMBOSIS OF THE INTERNAL CAROTID ARTERY

Report of Two Cases *

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Cerebrovascular accidents in the form of thrombosis are frequently seen by the practitioner. Over the past twenty-five years, several forms of therapy have been proposed, including vasodilators, stellate ganglion block, and anticoagulants. In the past five years, much attention has been focused on one group of patients who have suffered a "stroke," the cause of which is arteriosclerotic occlusion and thrombosis of the internal carotid artery. Although this cause for "strokes" has been recognized for many years, the diagnosis usually was not made before death, or, if recognized, the physician assumed a passive attitude towards its treatment. We now know that definitive treatment is available for such patients. The syndrome, diagnostic and confirmatory tests, and therapy will be discussed here briefly.

SIGNS AND SYMPTOMS

The clinical picture of thrombosis of the internal carotid artery is usually one of repeated minor cerebrovascular accidents, which previously had been considered as "cerebrovasospasms" or "little strokes" involving the terminal cerebral arteries. It is now recognized that stenosis of the carotid artery will give rise to the "little stroke syndrome." The patient's complaints may include a wide variety of symptoms, such as headache on the affected side, lightheadedness, personality changes, seizures, aphasia, fleeting disturbance of vision on the affected side, contralateral weakness, and incoordination or paresthesiae. Some believe that gradual stenosis of the internal carotid artery by advancing plaque formation will not give rise to these symptoms of cerebral ischemia because of the cerebral collateral circulation. It must be realized, however, that the state of the collateral circulation is the cardinal consideration, and when this is inadequate, or when the systolic blood pressure is low, a decreased flow of blood through the carotid artery will produce symptoms. Initially, symptoms tend to be intermittent, but as the occlusion progresses in the presence of an inadequate collateral circulation, they increase in frequency and intensity.

DIAGNOSIS

Proper treatment of these patients depends on the diagnosis being suspected during the stage in which the symptoms are transient. Aside from the history of minor "strokes" indicative of cerebrovascular insufficiency, suspicion is strengthened when pulsation in the internal carotid artery is diminished just below the angle of the jaw. It is important to realize that the area of maximal pulsation in the neck is at or just below the carotid bifurcation, which lies

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about one and one-half inches below the angle of the jaw. If the pulse is followed up to the angle of the jaw, it is probably the internal carotid artery that is being palpated. Infrequently, no difference in pulsation may be felt between the two sides in known cases of thrombosis of the internal carotid artery. It has also been suggested that the internal carotid pulse be sought intraorally along the posterior-lateral pharyngeal wall, but this method is often not easily tolerated by the patient. Measurement of the pressure in the central artery of the retina by means of an ophthalmodynometer is a useful and safe aid in the diagnosis of lesions of the carotid artery. The ophthalmic artery, the first and only extracranial branch of the internal carotid artery gives rise to the central artery of the retina, and a significant decrease in the pressure in this artery is a reliable indication of stenosis of the carotid artery or occlusion on that side.

If the clinical picture conforms to this description, regardless of a palpable difference between the internal carotid pulsations in the neck, the patient should be suspected of having an occlusion of the internal carotid artery. Not all patients, however, in whom this diagnosis is suspected are suitable candidates for surgical treatment. If the disease occurs in a person who has a reasonable chance of having segmental arteriosclerotic occlusion, then a definitive approach should be undertaken promptly and aggressively. The more localized and partial the obstruction, the higher the incidence of surgical success. The favorite site of occlusion is at the bifurcation of the common carotid artery into the internal and external carotid arteries. A carotid arteriogram can be carried out easily to demonstrate the occlusion.

TREATMENT

The methods of surgical treatment include: (1) arteriotomy with thrombo-endarterectomy, (2) bypassing the occlusion by means of a plastic vessel shunt from the aorta or subclavian artery to above the occluded segment, (3) resection of the occluded area with replacement by a plastic graft and reestablishment of arterial flow, (4) end-to-end anastomosis of a patent external or common carotid artery to the internal carotid artery above the occlusion. (Figure 1).

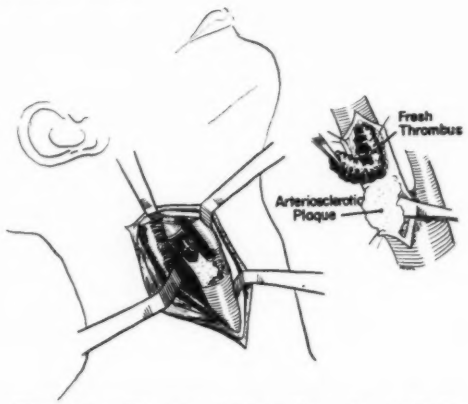

Figure 1.—Diagram illustrating the technique of carotid thromboendarterectomy.

The ideal candidate for surgical treatment has partial stenosis at the origin of the internal carotid artery which can be removed by thrombo-endarterectomy. By this technique, the vessel is incised longitudinally over the plaque (or in suitable instances where length of available vessel presents no problem, the artery is completly transected), and a plane is developed between the atheromatous plaque and the "normal" arterial wall. The occlusive debris is peeled out, after which the thinned vascular wall is reconstituted.

Operative success depends on both patency of the distal segment and extent of the occlusive process. Blood flow can almost always be reestablished successfully when the occlusion is partial, but the total occlusion must be submitted promptly to operation before the thrombus reaches the intracranial portion of the vessel. Unless retrograde bleeding occurs, it is likely that distal thrombosis or occlusive disease is advanced and blood flow cannot be reestablished. Occasionally, relatively fresh nonadherent distal thrombus can be extracted followed by brisk back-bleeding, with dramatic resolution of symptoms.

Recently, attention has been focused on coexistent stenosis at the origin of the vertebral artery, and further operative relief

of occlusion in this location is suggested. It may well be advisable in these instances of vertebral and carotid arterial thrombosis to use hypothermia if blood flow through the external carotid artery cannot be maintained during the procedure. When body temperature is lowered to 30 degrees C., cerebral metabolism is decreased, and function with longer periods of anoxia may be assured.

More frequent use of cerebral angiography will allow earlier diagnosis of cerebral ischemia due to occlusion of the carotid artery. Surgical treatment must be instituted promptly to prevent major cerebral infarction.

REPORT OF CASES

Case No. 1.—R. K., a fifty-one year old left-handed mill worker, was admitted to Touro Infirmary on June 26, 1958. For five days preceding hospitalization, he had noted intermittent right frontotemporal headache, moderate difficulty in verbal expression, light-headedness, moderate incoordination in the left arm and hand, and numbness and tingling in the left hand. On the morning of admission, he became drowsy and confused. On neurologic examination, he was observed to be drowsy and slightly confused. The cranial nerve segments were intact. Pulsations were diminished in the right internal carotid artery in the neck. The left upper extremity was weak, and movements of the left hand were poorly coordinated. The deep tendon reflexes were increased in the left upper extremity.

Within four hours of admission, a right percutaneous carotid angiogram was made with use of thiopental sodium. Complete occlusion of the right internal carotid artery was demonstrated just distal to the carotid bifurcation (Figure 2).

Within six hours of admission, a thrombo-endarterectomy was carried out on the right internal carotid artery with removal of an arteriosclerotic plaque and an associated fresh thrombus. Postoperatively, the patient received anticoagulants. He made an excellent recovery, and by the time of dismissal from the hospital on July 4, 1958, he was relatively asymptomatic. On neurologic examination at this time, only minimal residual weakness and slight incoordination in the left upper extremity were noted.

Case No. 2.—E. S., a forty-eight year old right-handed farmer, was admitted to Charity Hospital on May 26, 1958. Seven months before admission, he noted the onset of right frontal headaches and light-headedness. These symptoms recurred intermittently. One month before hospitalization, the headaches intensified, and he noted, in addition,

Figure 2.—Arteriogram demonstrating the complete occlusion of the internal carotid artery.

episodes of weakness and numbness of the left hand. On neurologic examination on admission, the patient was observed to be slightly confused, with minimal weakness and moderate incoordination of the left upper extremity. A neurologic survey was begun, but the patient deserted the hospital on June 11, 1958.

He was readmitted to Charity Hospital on June 24, 1958, with intensification of his previous complaints. On reexamination at that time, he was moderately confused, with left hemiparesis and impaired stereognostic sense in the left hand. The right internal carotid pulse in the neck was diminished. Three days after this admission, a right percutaneous carotid angiogram was made with use of thiopental sodium, and partial occlusion of the internal and external carotid arteries was demonstrated (Figure 3). On the same day, the

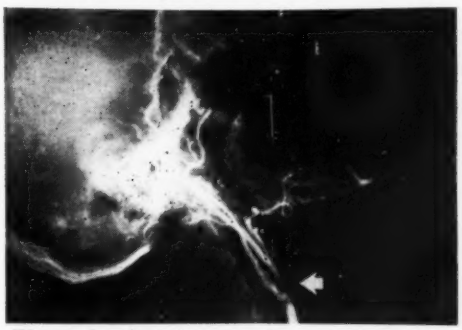

Figure 3.—Arteriogram demonstrating partial occlusion of the internal and external carotid arteries.

right internal carotid artery was explored, but the surgeon was unable to demonstrate adequately an occlusion. After the operation, the patient's left side became progressively weaker, and by the second post-operative day, he had left hemiplegia. A carotid angiogram was made again on July 9, 1958, and complete occlusion of the right internal carotid artery was seen at the carotid syphon. Further attempts at endarterectomy were not considered indicated.

ON THE CRITERIA FOR THE ADEQUATE MANAGEMENT OF DIABETES *

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The formulation of criteria for the adequate management of diabetes requires some consideration of the fundamentals of the disease. Diabetes is a disorder of intermediary metabolism involving fat and protein, as well as the more obvious carbohydrate disturbance. The generalized nature of the disorder might be anticipated from the concept of the metabolic pool,[1] wherein it is held that the enzymatic reactions through which metabolites are degraded, energy is produced, and tissue is synthesized, have in common at least one major pathway involving an abundant active metabolite.[2] This is a two-carbon fragment designated "active acetate," and has been identified by Lipmann et al,[3] as an acetylated coenzyme-A, one fragment of which is pantothenic acid, a vitamin. Insulin is notoriously involved in the pathologic physiology of diabetes. Its absence, relative or absolute, has been shown seriously to interfere, not only with the metabolism of carbohydrate, but also with that of fat and protein.[4] There is evidence in addition that in diabetes there is interference not only with the hexokinase reaction,[5] but also with the function of several vitamins which are components of enzyme systems concerned with intermediary metabolism.[6,7] The diabetic state is clearly related to insulin deficit although it is equally clear that other factors, especially hypothalamic, pituitary, and adrenal functions, are of great importance, though the relationship is not clear, and their role, whether primary or secondary, is uncertain. The adrenal relationship has been reviewed by Gordon.[8]

If these concepts are valid—and there is considerable supporting literature—it is to be anticipated that control of hyperglycemia, whether by dietary reduction of the metabolic load, insulin substitution therapy, or artificial stimulation of the insulin mechanism, cannot prevent all of the clinical effects of diabetes mellitus. That such therapy does fail to prevent all of the features of diabetes, though it has greatly improved the lot of the diabetic, is evident from the continued occurrence of the degenerative conditions commonly associated with diabetes of substantial duration. Nevertheless, until the ultimate molecular defect in diabetes, is fully characterized it appears reasonable to persevere in attempts at restoring to the norm as nearly as possible those deviations from the physiological which are definable and correctible with the knowledge and with the means at hand.

TWO PRINCIPAL PHILOSOPHIES OF MANAGEMENT

The fundamental elements in the present method of treatment are exercise, weight control, and insulin. The details and the precision of application of these elements vary largely between two schools of thought, although middle of the road voices, such as Leiter's are heard.[9] The one, which may best be termed the physiological school, exemplified by the Joslin group, attempts to rectify all deviations from the norm insofar as is humanly possible. The other, which may be termed the "free-diet" or clinical or practical school, exemplified by Tolstoi,[10] seeks to prevent symptoms and acidosis and to normalize the weight, insofar as is practical, with insulin enough to insure adequate carbohydrate utilization, and without undue inconvenience to the patient.

There are areas of agreement between the two schools. These are the provision of adequately nutritious diets, the use of enough insulin and the control of weight. While Tolstoi advocates a free diet, he does not advocate a licentious diet, and he does not call it a free diet but a "normal diet . . . suited to the patient's tastes, customs, and

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appetite. It does not mean that . . . he is to consume tremendous quantities of food or prodigious amounts of sweets. Furthermore, the maintenance of optimum weight is a must".[11] It is apparent that if the patient is to maintain "an optimum weight" he will have to exercise considerable restraint, unless his "tastes, customs and appetite" happen fortunately to coincide precisely with his metabolic needs.

Undoubtedly, the principle of insulin dosage enough to prevent symptoms and avert ketosis will in many instances coincide with the dosages required under strict control, remembering that Tolstoi's patients must maintain optimum weight, and this precludes overeating. But these are gross features of the pathophysiology of diabetes. The areas of disagreement are found in important details of management. The physiological school is concerned with the hyperglycemia, glycosuria and, at times, hyperlipemia [12] of diabetes, while the practical school is not. The physiological school feels that hyperglycemia is a major factor in the aggravation of the insulin deficit,[13] in the production of diabetic neuropathy, retinopathy, and nephropathy, and in the acceleration of the rate of development of atherosclerotic disease. The practical school believes to the contrary that hyperglycemia is not a factor, and that the degenerative aspects of diabetes are an integral part of the disease, and inevitable.[14,15] It should be noted, however, that Tolstoi does seek to control polyuria, and this necessarily involves a certain degree of control of glycosuria and of hyperglycemia. There is then a quite different philosophy of treatment between these two schools, and it is this difference which is crucial when the criteria of adequate management are to be selected.

It is conceded that incontrovertible statistics are not now available which could settle the issue finally because absolutely comparable series, matched in regard to age of onset, duration, uniformity of treatment, and type of diabetes, are not available. In particular, two major categories of diabetes mellitus are emphasized by Bornstein and Lawrence [16] and by Lawrence:[17] a severe type with usual onset at an early age, in which there is no available plasma-insulin, and in which an insulin antagonist may be present, and a mild diabetes, in which plasma-insulin activity is of the order of 70 per cent of normal, insulin insensitivity does not occur, and which is usually but not always found in the mature-onset diabetic. Pollack [6] suggests that severity of diabetes is definable in terms of longevity, deterioration of the individual, and association of degenerative changes; whereas others speak only in terms of insulin requirement. It is clear that series reported with mixed types and degrees of diabetes and with varying definitions of types cannot very well be compared.

PREVENTION OF DEGENERATIVE CHANGES

The pathogenesis of degenerative changes in diabetes has not been explained; this is also true of degenerative diseases in the nondiabetic. However, it is a fact that degenerative vascular disease in diabetes is not inevitable as is pointed up by the report by West [18] of normal renal and retinal findings after thirty-five years of diabetes. This gives hope that prevention may be possible. Root and Barclay,[19] in reviewing the Joslin series of 34,000 cases from 1898 to 1950, concluded that the disease shows a general tendency to progression but that a remarkable prolongation of life expectancy observed over the years could be attributed, aside from the advent of insulin, to early diagnosis and intelligent cooperation between the physician and the patient.

There is a high degree of association between obesity and diabetes, and between obesity in the diabetic and incidence of degenerative vascular disease. It is generally accepted, therefore, that ideal weights should be sought for the diabetic. All of us know, however, how difficult is the achievement of weight control. The stubborness of obesity is a continuing and frustrating problem. We are forced to admit that we have no really satisfactory understanding or treatment for it, and that starvation, while sometimes helpful, is no real solution.

Of interest in this connection is the apparent demonstration by Feller [20] that fat makes fat; that is, that adipose tissue can take up acetate, for example, and convert it into fat, and this at a rate faster than can liver tissue. Related to the problem of obesity is the question of fat content of the diet. Edeiken [21] in 1945 reported that low fat diets reduced incidence of premature cardiovascular abnormalities in younger diabetics. Malmros [22] suggested, from the lowered diabetic morbidity and mortality rates in Sweden during the World War II scarcity of butter-fat and eggs, that dietary fat may be a factor in diabetic vascular disease. Singh [23] reported amelioration of diabetes as regards insulin requirement and glucose tolerance curves in patients on a low fat diet; it is to be noted that his patients were also on an ideal caloric intake. Tuller et al [24] studying blood lipids in acidosis and coma found that abnormalities in serum lipids were extremely common. They cite the reports of Keiding et al [12] and Barach and Lowy,[25] indicating that generally poor control (in the Joslin sense) without actual acidosis, also causes abnormalities in the various lipoprotein bands. Hirsch, Phibbs, and Carbonaro,[25a] measuring esterified fatty acids, showed that postprandial hyperlipemia is markedly abrupt in diabetics, and, most importantly, that this rise is greater in the presence of hyperglycemia. This finding may not indeed be conclusive, but it cannot be denied that it represents a strongly suggestive link in the theory that seeks to relate hyperglycemia to disordered fat metabolism to vascular disease. It is suggested that these several reports indicate the desirability of considering, and in so far as possible, correcting those serum lipid deviations which may be encountered in the diabetic. It should be mentioned that Wilkinson [26] has suggested that the hypercholesterolemia of some diabetics could be a genetic phenomenon, inherited with diabetes merely because of the coincidental apposition of the corresponding genes in the same chromosome; this might vitiate a causal relationship between diabetes, its vascular complications, and hypercholesterolemia.

Duncan,[27] in discussing the favorable effects of exercise in diabetes, remarks that the possible beneficial effects of exercise in preventing atherosclerotic changes have been largely overlooked by the epidemiologic investigators in the field of atherogenesis versus dietary fat. Muri,[28] noting a relatively low incidence of retinal and renal changes among poorly controlled diabetics in a rural setting, characterized by social and financial stability on a simple plane, where heavy manual labor is the norm, suggested that a favorable environment, free of stress, with much physical exercise, might be the principal factor accounting for his findings. It is suggested that the degree of muscular development, and the physical "fitness" of the patient is worth noting in evaluating his management.

HYPERGLYCEMIA

Houssay [29] showed in 1947 that in experimental diabetes, hyperglycemia produces exhaustion of those beta cells which are damaged or reduced in number. Lukens and Dohan [30] found that minimizing hyperglycemia could arrest the diabetic state in pituitary diabetes in the cat; and more recently Lukens [31] showed that in the cat and dog massive glucose injections produced degenerative changes and atrophy of beta cells, and in some instances resulted in permanent diabetes.

Several ideas of the possible mechanism of production of degenerative vascular lesions by hyperglycemia have been advanced.

The most prevalent idea is that broad fluctuations in blood sugar level incite the stress reaction with consequent adrenocortical imbalance which, in turn, as suggested by Goodman,[32] affects the supporting fibrous connective tissue of smaller vessels, initiating the basic lesion. Kinsell [33] suggests that marked fluctuations of blood-sugar levels, by disturbing cellular osmotic relationships, damage the cells, especially those of the vascular intima, rendering them subject to abnormal deposition of lipids; any factor, endogenous or exogenous, which tends to increase plasma lipids

would enhance the process. This interesting hypothesis brings to mind that in the nondiabetic, blood sugar fluctuations (or other osmotic changes) though of lesser degree might also promote vascular damage, though more slowly. Such a view would correlate well with the observation that while degenerative vascular lesions occur in both nondiabetics and diabetics, they progress more rapidly in the latter. Givner [34] suggests that diabetic retinopathy may result from diabetes, B 12 deficiency induced by diabetes, and adrenal hyperfunction. It has been shown by Friedenwald [35] that cortisone injections in alloxanized animals resulted in renal lesions similar to intercapillary glomerulosclerosis, and retinal capillary lesions similar to those of the diabetic. Pollack [6] notes that in the diabetic there are nitrogen loss and disturbances in riboflavin and nicotinamide metabolism, and suggests that periods of intense glycosuria, which are often attended by mild ketosis, interfere with fundamental enzyme systems which are thought to be concerned in the pathogenesis of atherosclerosis. Wortham and Headstream [36] point out that in the strictly controlled diabetic nocturnal hypoglycemia and rebound hyperglycemia are apt to occur; and that in the loosely controlled patient changes from a higher to a lower blood sugar, constituting a state of relative hypoglycemia, tend to occur; and suggest that these fluctuations stimulate adrenocortical activity which may in accordance with the observations of Friedenwald and others, be instrumental in the production of the vascular lesions of diabetics. An editorial in the Lancet [37] summarizes this type of viewpoint in England: unstable or poorly controlled diabetes leads to stress, to repeated excesses of adrenocortical activity, and ultimately to the production of degenerative vascular disease. These concepts deserve continuing attention and may, in time, yield their own additions to the criteria of adequate management of diabetes. For the present it would seem desirable to obtain steroid excretion studies in carefully selected cases, such as progressive retinal disease and Kimmelstiel-Wilson syndrome.

CLINICAL REPORTS ON DIABETIC DEGENERATIVE DISEASE

Regarding the clinical reports of diabetic degenerative disease we find Larsson et al [38] reporting that the majority of a series of juveniles developed retinopathy between the tenth and fifteenth years of diabetes on a free diet regime, with blood sugar kept below 200 mg. per cent and urines kept free of ketone bodies. Dolger [15] reported retinal hemorrhages in every single case of a series of 200 after ten to fifteen years; his series included 55 juveniles about evenly distributed among good, fair, and poor control. He concluded that the degenerative changes of diabetes are an integral part of the disease, not a complication, and that duration of diabetes is the major factor in the incidence of these lesions. Zins [39] found evidence of intercapillary glomerulosclerosis in 82 per cent of diabetics autopsied; he states that it is found in all cases of greater than fifteen years' duration. He believes that the renal lesion will progress despite therapy. Stearns et al [40] in post-mortem injection studies of coronary vessels in diabetics found coronary arteriosclerosis in each of 50 cases; complete occlusions were found in 28 of these. These authors state that severity of coronary arteriosclerosis is related to duration of the disease; they observed no correlation with insulin requirement. Jackson et al [41] in a report of a ten years' study of 75 children on diets "adequate by accepted standards" and on regular insulin given in 3 or 4 doses daily, found that retinopathy developed in 46 per cent of the cases. They concluded that duration of the disease was the major factor but conceded that lack of control was also a factor in development of degenerative vascular lesions. This report is twice cited by Tolstoi [10,11] (who does not advocate regular insulin) as proving that good control is ineffectual in preventing vascular damage. It is worth noting, however, that only 4 per cent of these cases developed nephropathy. Kinsell [42] remarks that this regime involving multiple doses of regular insulin means giving insulin by test and that the blood sugars could not have been maintained in physiologic range. Pollack [6] has pointed out that regular in-

sulin alone is inadequate in that it does not permit a normal nitrogen balance in the diabetic. Beidleman and Duncan,[43] reporting a case of Charcot joint due to diabetic neuropathy, note that 23 such cases reported in the literature were found typically in patients with many years of poorly controlled diabetes. Leopold,[44] analyzing the ocular changes in a series of 100 diabetics on insulin and measured low fat, high carbohydrate diet, arrived at the following conclusions: Closely controlled management reduces the incidence of corneal wrinkles, complicated cataracts, and to some extent, that of deep retinal hemorrhages and exudates. Richardson and Bowie,[45] reporting on the same group, found that the diabetes worsened in 55 per cent, but inasmuch as diabetes characteristically worsens with time, it is to be noted that 45 per cent of the cases did not worsen. Again on the same series, Naide [46] concluded that adequately controlled cases had a lower incidence of peripheral vascular disease; however, premature peripheral vascular disease was uncommon, hence not a useful criterion. Wilson, Root, and Marble,[47] in reporting a series of young patients with diabetes of long duration, found that the well controlled cases had an incidence of 25 per cent serious degenerative lesions, against 93 per cent for the poorly controlled group. Similarly, controlled cases showed 63 per cent twenty-year survival against 38 per cent for the poorly controlled cases. Tolstoi,[10,14] asks with reference to this report, "why did 7 per cent on the "free diet" fail to develop serious lesions, while 25 per cent of the controlled group did develop degenerative lesions?" It were better to note that 75 per cent of the controlled, and only 7 per cent of the free diet group, escaped serious lesions. Tolstoi,[14] goes on to say, and I quote: "There is no question that the incidence of retinopathy was less in the chemically controlled group, what puzzles me is why it should occur at all." He continues, with what should be a solution to his puzzlement: "These investigators too claim that they do not attain ideal control, but only a better degree."

Keiding, Root and Marble,[48] in an analysis of 451 cases with onset under age 30 and duration ten to thirty-six years, found that vascular complications were materially less common in strictly controlled diabetes. These authors concede that duration of diabetes correlates directly with incidence of degenerative vascular disease, but state that this study shows that degree of control is of even greater importance; they add that even excellent control according to their standards is far from ideal control, so that present methods cannot be expected to yield complete prevention of complications.

It remains to be seen whether tolbutamide will enhance the excellence of control, though it is of course not useful in the growth onset type of diabetes. To Keidling's comments should be added that it is well to remember that nondiabetics also develop vascular disease, and that unless diabetes should prove to hold the key to the general problem of atherosclerosis, we can scarcely expect control of diabetes, however perfect, to prevent vascular disease completely. The findings of Keidling, Root and Marble are reasserted by Marble,[49] with special reference to coronary artery disease, and concurred in by Sheppard [50] with special reference to ocular changes.

From England, Dunlop,[51] a convert to the physiological school, reports that in his cases the poorly controlled ones showed degenerative vascular changes and neuropathy three times more frequently than the well controlled; and Matthews,[52] reporting on 545 cases, found an incidence of retinopathy and albuminuria three times greater in the poor-control group, as compared to the fair-control group; he found this to be true in all age groups and durations of diabetes. On the continent Constam et al [53] report an incidence of 83 per cent severe degenerative vascular complications in a poorly controlled group, and 42 per cent in the combined good and fair control group of a series of cases of twenty or more years duration; and Fanconi [54] reports that in his experience, the well controlled patients show a lower incidence of late complications. This author makes the interesting suggestion that in the well-controlled diabetics, care for their health and avoidance

of excesses may be key factors in lowered incidence of late complications.

Joslin,[55] in commenting on the characteristics of diabetics awarded the "Quarter Century Victory Medal," states that, "searching for light on treatment it is evident that these patients, particularly the younger ones, went through a Spartan regime during the first few years they had the disease." Elsewhere,[56] he deplores, however, the overall incidence of 85 per cent complications after fifteen years of diabetes, but finds grounds for some optimism in the fact that one in ten diabetics can expect to live longer with diabetes than without it. A summary viewpoint on the question of control of hyperglycemia in diabetes is given by Colwell,[57] who states that, "We should maintain normal levels of blood sugar in an effort to avoid vascular complications unless and until it is proved unnecessary." It is well to reiterate that no one disputes the desirability, in the case of known or suspected coronary insufficiency, of allowing a more liberal type of management, appreciating that for such a patient the present and real dangers of hypoglycemia outweigh the long range dangers of hyperglycemia.

OUTLINE OF CRITERIA FOR ADEQUACY OF MANAGEMENT

With the foregoing considerations in mind, an outline of criteria for assessing the adequacy of management of diabetes is proposed as follows:

1. The patient should be kept free of symptoms and possessed of a sense of well-being.

2. He should be maintained at the ideal weight for his height and build, on a low-fat, high-protein, normal carbohydrate diet.

3. Insulin should be used in such dosage, and of such type, and timing, as to procure normoglycemia throughout the twenty-four hours; tolbutamide should be reserved for mild, mature-onset cases that cannot be controlled on diet alone.

4. He should be required to adhere to a program of physical fitness, even bordering on the athletic, with maximal activity within his limitations.

5. He should test his urine frequently, even three times daily in many cases, accepting the test as a natural part of his daily routine, even as he accepts polishing his shoes, combing his hair, and so on. There should be minimal or slight glycosuria but rarely, except in the coronary cases whose test should almost always show a trace.

6. A blood sugar should be done two or three times a year in the average case, more often in the labile and in the presence of coronary insufficiency. A nocturnal blood sugar should be checked occasionally in coronary cases.

7. Serum cholesterol and other lipids should be checked from time to time, and corrected whenever possible; the cholesterol should be kept below 200 if possible.

8. The patient should be reasonably free of emotional problems through whatever means, medicinal, social planning, recreational, psychotherapeutic, found indicated.

9. In selected cases, with lability, or associated with progressive retinal or renal disease, steroid excretion studies should be considered. General endocrine work-up may at times be indicated.

These criteria no doubt may be difficult of achieving in some cases, and naturally due regard must be had for the possibility of burdening the patient beyond his capacity. Nevertheless, so long as there is some reason to believe that maintaining the patient's homeostasis might protect him from degenerative changes, and until proof to the contrary, or a general solution to the problem of degenerative vascular disease is found, this writer believes that criteria such as are here presented, are rational and worth the effort and patience required.

SUMMARY

1. Some theoretical considerations on the nature of diabetes and its relation to degenerative disease are presented.

2. The two principal philosophies of management are discussed.

3. Experimental and clinical findings suggesting a relationship between diabetes, its chemical manifestations, and its associated degenerative vascular disorders are discussed.

4. Criteria for the adequate management of diabetes are proposed.

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PULMONARY EMBOLISM AND INFARCTION *

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The most common pulmonary disease encountered in general hospitals, in recent years, is pulmonary embolism. This conclusion was reached by two different groups of observers, one in the United States and the other in England. Each group set out to determine the incidence of pulmonary embolism in general hospital practice and trained their staff to be especially alert for the various manifestations of this disease. As a result, it was shown at the Graduate Hospital, University of Pennsylvania, that pulmonary embolism was more common than either bronchogenic carcinoma or pneumonia during the eighteen month period of study from 1955 to 1957.[1] Short made similar observations on a general hospital population in Bristol, England, during the years 1947 through 1950, and reached the same conclusion concerning pulmonary embolism.[2]

When viewed from the standpoint of autopsy material in general hospitals, the incidence of pulmonary embolism has remained remarkably constant at 10 per cent over a period of years. This figure is even higher in necropsies on inmates of nursing homes in large cities, reaching 25 per cent.[3] Thus it is evident that from both the clinical and pathological viewpoints, this condition is relatively common.

Although the two terms, pulmonary embolism and infarction are sometimes used interchangeably, they are by no means synonymous, and neither do the two conditions always coexist. Pulmonary embolism indicates the presence of bland emboli lodging in and occluding one or several branches of the pulmonary arterial tree. This condition is followed, in 50 to 75 per cent of cases, by pulmonary infarction, which refers to necrosis of the pulmonary parenchyma distal to the occluded artery. Typically pulmonary infarcts are hemorrhagic, and the area of necrosis is packed with extravasated erythrocytes, which may come either from the bronchial arterial supply of the lungs or from collateral pulmonary capillary anastomoses.[4] Infarction of lung tissue following embolism is not likely to occur unless there is antecedent embarrassment of the pulmonary circulation, as in chronic passive congestion, or unless there is coexistent parenchymal disease, such as pneumonia or bronchiectasis.

It has been estimated that for every fatal case of pulmonary embolism, approximately 9 or 10 nonfatal cases should be diagnosed clinically if the visiting staff is alert to the possibility.[5] This favorable ratio of 10 nonfatal to 1 fatal case is prevalent in very few general hospitals in this country at present, but it is a goal to keep in mind.

Although it was formerly believed that pulmonary embolism was usually seen only in postoperative or postpartal cases, it has become increasingly more evident that it is commonly encountered in medical cases as well, particularly in patients with cardiovascular disease.[6] Sixty per cent of all cases of pulmonary embolism occur in medical patients and at least half of these patients have heart disease. The remaining 40 per cent occur in surgical and obstetrical patients.[7]

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A study of *autopsy* material from Mercy Hospital, a 200 bed general hospital in New Orleans, revealed that pulmonary embolism and infarction was the direct cause of death in 32 cases over the five year period 1953 through 1957. During this same period of time there were 266 autopsies performed, so that the incidence of pulmonary embolism and infarction was 12 per cent of all autopsy cases. An analysis of the type of patient in which this complication occurred and the type of surgical procedure or medical disease preceding embolism is shown in Tables 1 and 2.

TABLE 1
TYPE OF CASE

	No. of Cases	Per Cent
Medical	19	58
Surgical	8	25
Obstetrical (amniotic fluid emboli)	2	7
Ambulatory	2	7
Post-traumatic	1	3
Total	32	100

Mercy Hospital in New Orleans 1953 - 1957, 266 autopsies, 32 cases of pulmonary embolism and infarction.

TABLE 2
SURGICAL OPERATIONS

Cholecystectomy		3
Craniotomy		1
Carcinoma of Rectosigmoid (exploratory laparotomy)		1
Ileocolostomy		1
Pneumonectomy		1
Tooth Extraction		1
UNDERLYING MEDICAL DISEASE		
Carcinoma		6
Pancreas	1	
Breast	1	
Gall Bladder	1	
Liver	1	
Lung	1	
Ovary	1	
Myocardial Infarction		5
Congestive Heart Failure		4
Acute Pancreatitis		1
Hydronephrosis and Bilateral Hydroureter		1

ETIOLOGY AND PATHOGENESIS

The source of pulmonary emboli in 85 to 95 per cent of cases is thrombosis of the deep veins of one or both lower extremities.[7,8] The thrombotic process usually begins in the plantar or calf veins and then propagates proximally along the deep venous tributaries and may extend to the iliofemoral system. A thrombus in the right side of the heart may produce pulmonary embolism. Occasionally the prostatic, pelvic and hemorrhoidal veins may be the source of emboli, and even less common are the veins of the upper extremity. However, it should be remembered that traumatic phlebitis of the antecubital veins secondary to repeated venipunctures, prolonged insertion of polyethylene tubes, and such intravenous medications as hypertonic glucose or saline, or bromsulfalein, may serve as the site of origin of pulmonary emboli.

There are certain conditions which predispose to thromboembolism, and knowledge of their existence is of significance in leading to the suspicion of pulmonary embolism. These are obesity, cardiac disease (especially congestive heart failure), anemia, varicose veins, previous thrombophlebitis, severe infections and malignancies. Advanced age, especially beyond 50 years, is a frequent predisposing factor. At least half of the cases are over 50 years of age. Trauma to the lower extremities, with and without fracture, prolonged immobilization and certain hematalogic disorders (e. g. polycythemia vera) also act as predisposing causes. In postoperative cases, embolism occurs most often from the eighth to the fourteenth postoperative day, although 25 per cent occur during the first week and 25 per cent after the second week. The incidence of embolism is highest in the more severe and extensive surgical procedures, such as gastrectomy, cholecystectomy, splenectomy and hysterectomy.[9]

Massive pulmonary embolism which occludes the main pulmonary artery and proves fatal in almost all cases, is not associated with infarction of lung tissue. The blockage in the lesser circulation produces increased pressure proximal to the site of obstruction with resultant strain on the right ventricle which in turn causes electrocardiographic and roentgenologic changes. The failure of the left ventricle to receive blood from the lungs results in decreased systemic output, fall in systemic blood pressure, and shock.

Pulmonary infarction, whenever it occurs, is located in most instances in association with a pleural surface, either peripheral or interlobar and mainly in the lower lobes, more often in the right lung. Infarction usually extends to the surface of the lung to involve the pleura, which reacts by an outpouring of fluid, either serous or serosanguinous. Pleural effusion is thus a frequent finding. Blood exudes into the alveolar spaces, and hemoptysis may occur also.

Chronic cor pulmonale may result from repeated bouts of pulmonary infarction or from recurrent showers of small emboli, occurring over a period of several years and producing right ventricular hypertrophy and eventually right sided heart failure. These showers of small emboli to the tertiary branches of the pulmonary arterial tree do not produce infarction.

SYMPTOMATOLOGY

The clinical syndromes produced by pulmonary embolism and infarction may be broadly divided into three groups: (1) Massive pulmonary embolism which produces a rather typical picture, and in most cases can be diagnosed by clinical, roentgenological, and electrocardiographic methods. It is due to complete or almost complete obstruction of the main pulmonary artery and results in the acute cor pulmonale described by Mc Ginn and White.[10] (2) Single or multiple pulmonary infarcts occurring close together in time, whose clinical manifestations are protean, and which are most likely to be confused with pneumonia, pleurisy with or without effusion, myocardial infarction, congestive heart failure, or tuberculosis. (3) Recurrent multiple small pulmonary emboli over a long period of time (several years) which give rise to pulmonary hypertension and chronic cor pulmonale. The last clinical syndrome is a rather rare occurrence, that of massive pulmonary embolism is more common and the syndrome of single or multiple pulmonary infarction is the most frequent of all. This classification is devised to depict the many and varied manifestations of pulmonary embolism and to give a broad panoramic view of its multiple aspects.

Massive pulmonary embolism is characterized by the sudden onset of agonizing dyspnea which remains throughout its course the predominating symptom and is out of all proportion to other findings. The next most common symptom is substernal oppression which often leads to the mistaken diagnosis of myocardial infarction. Vasomotor shock usually occurs and the patient's skin is pale, cold and clammy with distention of the cervical veins and constriction of the peripheral veins. The physical findings consist of apprehension and a feeling of impending doom, which very often the patient is unable to describe but is quite evident to the observer by facial expression. There is increased prominence and pulsation noted by inspection and palpation in the region of the 2nd and 3rd intercostal spaces just to the left of the sternum. A loud systolic pulmonic murmur is usually heard and the pulmonic second sound is sharp and increased in intensity. A friction rub may be heard in the same region. Diastolic gallop rhythm is sometimes present. There are dilation and increased pulsation of the jugular veins, and cyanosis is usually present. Fever may rise as high as 104°F, and a moderate leucoytosis is usually present. The chest roentgenogram reveals no evidence of infarction, but the main pulmonary artery region is prominent and decreases in size if recovery ensues. The electrocardiogram gives great assistance and is usually quite typical. The changes consist of a prominent S wave in Lead I with a Q wave in Lead III, depressed RS-T_1 elevated RS-T_3 and inverted T in Lead III, and in some cases inverted T waves in the right sided precordial leads.

The cardiovascular signs may subside quickly in hours or days until death or recovery occurs.

There is a special type of massive pulmonary embolism which may occur in obstetrical patients. It is due to amniotic fluid emboli and should be mentioned briefly. This catastrophe is ushered in by violent uterine contractions and premature rupture of the fetal membranes after which the patient develops sudden severe dyspnea,

cyanosis, a shock-like state, and usually a fatal termination.[11]

The second clinical syndrome of single or multiple pulmonary infarcts may vary in severity from mild to overwhelming. The typical picture of chest pain, hemoptysis, and x-ray density in the chest film is present in less than 50 per cent of cases, so that reliance on the presence of all these signs is unsound in making the diagnosis or instituting treatment. Pulmonary embolism with infarction may assume any of four guises: (1) pulmonary, (2) cardiac, (3) abdominal, (4) neurologic.[1]

Among the pulmonary diseases it may resemble are pneumonia, pleurisy (dry or with effusion), lung abscess, tuberculosis and lung cancer. Resemblance to pneumonia is more often close and it is frequently misdiagnosed as such. But lack of preceding respiratory tract infection and recurrent episodes in one lung and then the other differentiate pulmonary infarction. Pulmonary infarction is often the cause of dry pleurisy, and when effusion develops it is grossly bloody in 50 per cent of cases, mainly those with congestive failure. The rounded shadow of an infarct on the chest film may be mistaken for the solitary nodule of lung cancer. Pulmonary edema in the same or opposite lung may result from pulmonary infarction.

The cardiac syndromes it may mimic are myocardial infarction, coronary insufficiency, congestive heart failure and chronic cor pulmonale. Recurrent episodes of chest pain in patients recovering from myocardial infarction without further electrocardiographic evidence of new or spread of the old infarction are often due to repeated pulmonary embolism and infarction. Not only may pulmonary embolism be a sequel of myocardial infarction, but the converse may be true and myocardial infarction or coronary insufficiency result from severe pulmonary embolism. Congestive failure predisposes to pulmonary embolism and the latter, in turn, commonly precipitates heart failure or causes it to persist despite therapy. Pulmonary embolism may produce arrhythmias, usually paroxysmal and quite often atrial fibrillation.

Syncope associated with chest pain and dyspnea may be the first manifestation of pulmonary embolism and infarction. Occasionally syncope may occur alone. Hemiplegia and convulsions may result from decreased cardiac output leading to cerebral anoxemia following pulmonary embolism.

Occasionally pulmonary embolism may mimic the acute surgical abdomen, acute cholecystitis or subphrenic abscess.

The symptoms of pulmonary embolism with infarction are sudden dyspnea, chest pain which may be pleuritic or anginal in nature, shock-like state, with a feeling of faintness (and occasionally syncope), restlessness and sweating. Cough is a common symptom but hemoptysis is seen in only one-third of the cases of infarction. Bloody sputum is considered rather typical of pulmonary infarction and hemoptysis may be quite copious. The physical signs in order of importance are: tachycardia (up to 150/min.) out of all proportion to the fever and which is usually not affected by digitalization; tachypnea; cyanosis. Pleural rub, localized crepitant rales and decreased breath sounds are important local signs, and if present, aid greatly in the diagnosis. Exquisite tenderness over the site of the chest pain has been described as characteristic of infarction. Fever, usually mild, is present, but may be as high as 103 or 104°F. Jaundice is rather uncommon, but is important, if present. Examination of the legs is likely to be more informative than examination of the chest. Pain on direct posteroanterior compression of the calves is the most sensitive indicator of thrombosis of the deep calf veins. Other leg signs, including sole, calf or thigh tenderness, swelling and Homan's sign (calf pain on dorsiflexion of the foot) are found in less than half the cases, but should be diligently sought for daily.

The roentgen ray examination of the chest is the most valuable of all laboratory procedures. At the onset and for twenty-four hours thereafter, there may be merely slight elevation of the hemidiaphragm on the affected side. A negative chest film,

especially during the first two days, does not rule out the diagnosis of pulmonary embolism. If infarction ensues, one or more areas of opacification appear, usually in the lower lobes and more often the right than the left. These are usually hazy and poorly defined with indistinct margins, are rarely triangular but are usually round, oval or irregular. They occur nearly always in relationship to a pleural surface and particularly where two pleural surfaces meet. The cardiac or central margin is convex or "humped".[7] With organization and healing of the infarct, which usually takes two weeks, the involved area becomes converted into a distinct and sharply defined linear scar. If effusion develops, the infarct may be obscured by it, or by an enlarged heart, or it may be buried in the costophrenic angles. With incomplete infarction, which when it occurs, does so in patients with normal lungs, the roentgen ray shadows last only two to three days and heal by resolution and so leave no residual scarring.[12]

The electrocardiogram may manifest one or more of a variety of abnormalities, of a transient and characteristic nature, whose demonstration in a patient with an appropriate clinical picture supports the diagnosis of pulmonary embolism. There may be present one or more of the components of the full acute cor pulmonale pattern, previously described. Or there may be the electrocardiographic findings of "coronary insufficiency," manifested by ST segment depressions and T wave inversions over the left precordial leads. Again there may occur transient inversion of T waves in the right precordial leads. Arrhythmias are common, and atrial flutter or fibrillation occurs in as high as one-third of the cases, and is usually paroxysmal.[1,13]

The laboratory findings are a mild to moderate leucocytosis, elevated sedimentation rate, and elevated serum bilirubin. The most recent test to be introduced, and of special value in differentiating pulmonary and myocardial infarction, is the serum glutamic oxalacetic transaminase (SGO-T) level. This determination is elevated (over 40 U/ml.) early in myocardial infarction (in twenty-four to forty-eight hours) but is normal in uncomplicated pulmonary embolism and infarction. When pulmonary embolism is complicated by hepatic or biliary disease, myocardial infarction or musculoskeletal diseases, the SGO-T level is usually elevated, and so becomes unreliable as a differential test.[1,14]

The third clinical syndrome of multiple small pulmonary emboli to the smaller (tertiary) pulmonary arteries over a period of years results in pulmonary hypertension and chronic cor pulmonale. Owen and associates [15] collected twelve such cases of "silent" pulmonary emobli, and the clinical picture was usually that of increasingly severe congestive heart failure of undetermined etiology. Dyspnea and cough were frequent symptoms. Common auscultatory findings were accentuation of the pulmonic second sound, gallop rhythm and harsh systolic murmurs along the left sternal border in the 3rd and 4th interspaces. X-ray studies showed prominence of the main pulmonary vessels and right ventricular hypertrophy. All patients expired because of intractable congestive heart failure.

DIAGNOSIS

The diagnosis of pulmonary embolism and infarction is essentially a clinical one as there is no single symptom, sign, or laboratory finding which is pathognomonic of this condition. Since the diagnosis in the living patient can never be absolute, it is often difficult to convince the skeptical. The greatest error in recognition results from demanding too much to establish the diagnosis. The so-called typical picture with chest pain, hemoptysis, x-ray opacity, and leg signs occurs in less than half the cases, and to await the appearance of these is to miss the opportunity for early therapy. So first and foremost must be a *high index of suspicion of this condition* in all patients who have (1) the proper historical background, (i. e., postoperative, postpartal, post-traumatic, *bedridden patients and especially cardiacs)*, and (2) one or more symptoms or signs of the pulmonary, cardiac, abdominal or neurological syndromes previously mentioned.

It is most commonly mistaken for pneumonia, either viral, or bacterial which is slow to respond to antibiotics. But the recurrent nature of pulmonary embolism is one of its most distinctive features, and involvement of now one lung, and then the opposite one is strong evidence in its favor. This point is worthy of note in differentiating it from pneumonia or myocardial infarction as neither one of these latter conditions recurs rapidly every few days.

Other clues to keep in mind are an unexplained fever, increased pulse and respiratory rates, especially in a bed patient; abrupt onset of dyspnea, associated with tachycardia and tachypnea out of proportion to the fever; the finding of localized chest tenderness or leg signs; recurrent episodes of syncope, especially when associated with dyspnea or chest pain or both; episodes of weakness and sweating; paroxysmal atrial fibrillation; refractory heart failure; repeated attacks of what are thought to be coronary occlusion occurring close together. And then, as a last point, the most common cause of fever or jaundice or both in a patient with congestive heart failure is pulmonary infarction.

TREATMENT

The prevention of thrombosis of the deep leg veins is the fundamental problem and is more important than its treatment. At the present time there are a number of measures employed to this end, and it is not definitely known which is the most effective. Any procedure or position conducive to blocking of the pelvic or leg veins should be avoided. Deep breathing exercises, active and passive leg exercises and early ambulation all benefit the circulation by preventing stasis and are of value. It must be remembered that early ambulation means that the patient be up on his feet walking (with assistance if necessary) as soon as possible, and not seated on the edge of bed with legs and feet dangling, as this actually impedes the venous return and encourages thrombosis. The wearing of knee-length elastic stockings routinely in all bed confined patients (medical and surgical) has been shown by Wilkins and Stanton [16] to reduce appreciably the incidence of fatal pulmonary embolism. The compression afforded by the stockings increases the linear velocity of blood flow in the venous channels and in this manner aids in preventing venous thrombosis. These measures are advisable in all postoperative, postpartal and bed-confined medical cases. The routine use of venous ligation or anticoagulants as a prophylactic measure in those patients undergoing surgery is open to question and not widely practiced. However, all cases prone to develop thromboembolic episodes should be treated with either ligation or anticoagulants. This would particularly apply to patients with acute myocardial infarction and those in congestive heart failure, confined to bed.

The treatment of pulmonary embolism and infarction is directed at (1) the emergency treatment of severe pulmonary embolism, (2) the relief of symptoms due to infarction, and (3) the prevention of further embolization.

The first few hours following severe pulmonary embolism are critical. There is widespread reflex contraction of the pulmonary vascular bed as a result of the circulatory reaction to embolism. Atropine sulfate intravenously in large doses (gr.-1/60) has been advocated to block vagal reflexes. Papaverine intravenously (gr. ½ to 1) alone or combined with atropine has a beneficial effect on dyspnea and is a pulmonary vasodilator. Oxygen by tent or mask in high concentration is of great value for the dyspnea and cyanosis, and morphine or meperidine (Demerol) is useful for its sedative and analgesic effect. The patient should be at absolute bed rest and instructed to avoid deep breathing, violent coughing and straining at stool in the hope of preventing another attack.

Pulmonary infarction often causes severe pleuritic pain which may be difficult to relieve. Paravertebral procaine block or local intercostal procaine infiltration affords the best relief. Codeine or meperidine (Demerol) may be used in small doses to avoid respiratory depression. Sedative cough preparations are given for hemoptysis, which at times may be profuse; anticoagulants are not contraindicated in the

presence of hemoptysis.[17] Administration of antibiotics is justified since infarcts occasionally become infected and progress to abscess formation.

The prevention of further embolization may be effected by venous ligation or anticoagulant therapy and the latter is the preferred method in most cases. Unless there is some contraindication, anticoagulant therapy should be initiated immediately and should consist of heparin alone or combined with either dicumarol or phenindione (Hedulin). The contraindications to anticoagulants are liver disease, renal insufficiency, bleeding diatheses, active peptic ulcer and ulcerative colitis. Heparin when used alone is probably best given as intermittent intravenous injections of 100 mg. at four to eight hour intervals the first day, and 75 mg. at eight hour intervals for the next two to three days. The patient is then ambulated. It is continued at 75 mg. at twelve hour intervals for two days, then daily for two succeeding days.[18] Protamine or fresh blood transfusion will combat bleeding from heparin, which is rare.

More commonly, heparin is given in combination with dicumarol or Hedulin, and then heparin is used for the first two to three days for its immediate anticoagulant effect and dicumarol or Hedulin is given simultaneously and continued for two to four weeks or until the patient is fully ambulatory. Both dicumarol and Hedulin have the advantage of oral administration, but Hedulin acts more rapidly (eighteen to twenty-four hours), produces a more uniform reduction in prothrombin concentration, and thus abolishes daily prothrombin determinations. The initial dose of Hedulin is 200 to 300 mg., in 2 equal doses twelve hours apart the first day and thereafter a daily maintenance dose of 50 to 100 mg. in 2 equal doses. Prothrombin determinations are done daily for the first three days. Thereafter they are done every seven to fourteen days, depending on the patient's response.

Although bilateral superficial femoral vein ligation was introduced several years before the anticoagulants it has became less popular recently, partly because of the frequency of subsequent edema, but, more important because it too often fails to accomplish its purpose. Anlyan and his associates [19] have pointed out that quite often there is thrombus formation proximal to the site of ligation with resultant embolization. This occurred in 5 of 7 patients of theirs treated with ligation and one of these died of fatal pulmonary embolism. These authors feel that if anticoagulant therapy is contradicted the inferior vena cava should be ligated.

Venous interruption, however, still has a place in the therapy of pulmonary embolism, and is employed whenever anticoagulants are contraindicated, or whenever they fail to control embolization. Surgical ligation usually signifies *bilateral superficial femoral vein ligation* at a point immediately distal to the profunda femoris vein. If thrombosis extends proximal to this site, then inferior vena cava ligation just proximal to the junction of the two common iliac veins is done. It is felt by some that ligation of the superficial femoral veins is becoming an unsound measure in ligation therapy [19,20] and that inferior vena cava ligation is a more suitable procedure. In certain conditions producing pulmonary embolism, ligation of the inferior vena cava is the procedure of choice, namely: postpartal period, suppurative pelvic thrombophlebitis, postoperative prostatectomy, combined abdomino-perineal resection.[21] Femoral vein ligation, in these circumstances, is obviously not feasible as the source of emboli is in the pelvic veins which empty into the inferior vena cava proximal to the site of ligation.

Pain and edema of both lower extremities occur quite frequently following inferior vena cava ligation. Long term care to combat these distressing symptoms consists of periodic leg exercises, elevation of the lower extremities at night, wrapping of legs and thighs during the day (with elastic bandages) and avoidance of a static dependent position for the lower extremities.[21,22] A recent adjunct to our armamentarium in the relief of pain, fever, and edema following inferior vena cava ligation is phenylbutazone (Butazolidine). This is

administered for five to seven days, 200 mg. three times daily for the first three days, then 100 mg. three times daily for the next two to four days. Symptoms of edema, pain, fever, and local tenderness begin to subside in two days.[23]

CASE REPORTS

The following case summaries illustrate the many aspects of pulmonary embolism and infarction:

Case No. 1: A. DB., 60 year old white male. February 1, 1958 - February 1, 1958. Patient had "flu" two weeks prior to admission, recovered and had relapse five days later. Been in bed since then. Has had pain and tenderness in right calf the past five months. At 2 A.M. on February 1, 1958, woke up feeling very weak and with a cold sweat, substernal discomfort and severe, acute dyspnea which was the predominant symptom.

Examination — Blood Pressure: 70/40; Pulse: 145-152/m (regular and feeble). Skin cold and clammy, ashen gray. Obviously dyspneic with apprehensive look. Heart tones distant with rapid, regular rhythm. P_2 equals A_2. Lungs—anteriorly and laterally normal breath sounds. Severe tenderness of right calf. Electrocardiogram: (Figure 1) deep S_1, Q_3 T_3 pattern of pulmonary embolism. Expired twelve hours after onset.

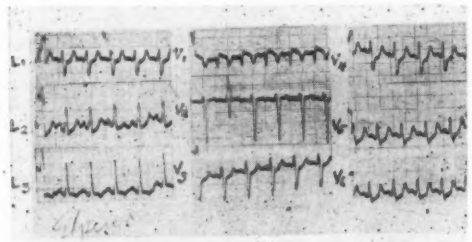

Figure 1.—(Case No. 1, A. D. B.) Electrocardiogram typical of massive pulmonary embolism. Note deep S_1, presence of Q_2 and Q_3 and inverted T_3, depressed ST segments in V_3, V_4, V_5, and V_6.

Comment: This patient had deep vein thrombosis of right calf for five months preceding fatal episode. He received no therapy for it. The first bout of pulmonary embolism was the fatal one. The symptom of agonizing dyspnea overshadowing chest pain is characteristic, although his family physician felt positive he had a myocardial infarction when he first saw him at home. The electrocardiogram is typical of massive pulmonary embolism and has significant differences from that of myocardial infarction, namely deep S_1, associated with Q_3 and inverted T_3, and change in the electrical position of the heart (Vertical shift).

Case No. 2: A. J. M., 16 year old white male—November 1, 1953, to November 10, 1953. Patient was in an auto accident four days prior to admission and injured his right knee. His right foot got cold and swollen and his right knee became painful. He developed tightness in the chest one day prior to admission, and was admitted for severe dyspnea, severe substernal tightness, and inability to move arms. These symptoms occurred suddenly at 8 P.M. on the day of admission. He collapsed and was taken to the hospital and given artificial respiration on the way. His blood pressure was 0/0; pulse unobtainable; heart tones distant; lungs clear. Begun on oxygen, papaverine and atropine, intravenously, and anticoagulants (heparin and dicumarol).

A chest film revealed prominence of the main pulmonary artery which diminished to normal size 5 days later. Electrocardiogram revealed deep S_1 Q_3 and inverted T_3. Second small embolus with collapse on November 3, 1953. At that time his lungs were clear, heart sounds normal except P_2 greater than A_2. Bilateral superficial femoral vein ligation was done and his pulse rate dropped from 100 to 60/m. He was discharged on November 10, 1953, and anticoagulants were continued for two weeks. At the present time the patient is well and at work.

Comment: This patient was rather young for massive pulmonary embolism and it resulted from a relatively minor traumatic episode. He had a second embolus two days after the first despite anticoagulants, and bilateral femoral vein ligation was done. He recovered and is well at present.

Case No. 3: J. D. K., 85 year old white female, January 20, 1958, to January 21, 1958. Patient was in a nursing home for several months, and had pernicious anemia which was under control with Vitamin B_{12} injections. Her complaints were lack of appetite, dyspnea, and change in general condition. Her blood pressure was 90/0; pulse irregular and weak, 110/m. Chest kyphotic. Heart sounds were distant with occasional premature contraction. Lungs revealed dullness on percussion at the right base, with fine and medium, moist rales at the right base anteriorly, laterally and posteriorly, with decreased breath sounds in the same area.

White blood count 18,800; 89% Neutrophils; Blood Urea Nitrogen 66 mg. %; Electrocardiogram revealed left ventricular hypertrophy; EPA of chest revealed infiltration throughout the right lung field. (Figure 2)

Patient expired on January 21, 1958, the day after admission.

Comment: This was an elderly female

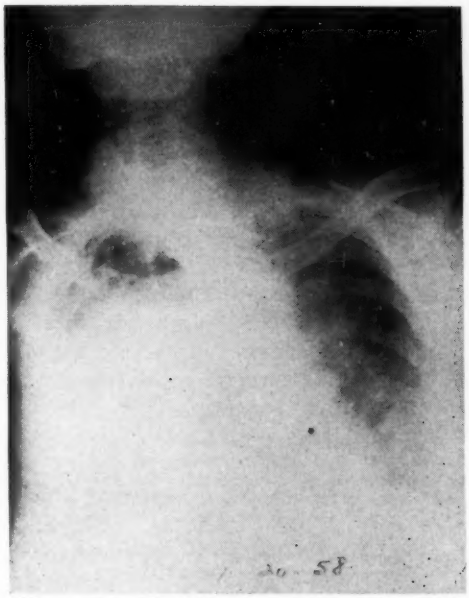

Figure 2.—(Case No. 3, J. D. K.) This patient at autopsy had bilateral pleural effusion and multiple pulmonary infarcts throughout right lung.

living in a nursing home. She was very inactive most of the time and usually sat in a rocking chair all day. She gradually deteriorated as a result of multiple pulmonary infarcts and expired one day after admission to the hospital. There were multiple infarcts in the right lung with a right pleural effusion and pulmonary edema of the left lung.

Case No. 4: M. B., 68 year old white female, January 31, 1958, to March 23, 1958. Patient was admitted because of progressively severe dyspnea at rest and after exertion, of one to two weeks' duration. She had also been complaining of severe leg pains on exertion at the beginning, but later at rest also for the past few months. She has had hypertension of many years and also angina pectoris.

Her blood pressure varied from 125/70 to 180/110, and pulse 64/m. She had no cervical vein distention. Her heart was enlarged to the left with regular rhythm and apical grade II systolic blowing murmur. No palpable liver. Minimal pitting edema of the legs with absent dorsalis pedis and posterior tibial pulsations bilaterally. Calf tenderness was present bilaterally and was more marked on the left side.

She was digitalized with no improvement, and during the first week in the hospital, she experienced three bouts of syncope. The dyspnea continued unabated and she developed left anterior chest pain with localized rales over the same area.

X-ray film (Figure 3) revealed fluid in the left

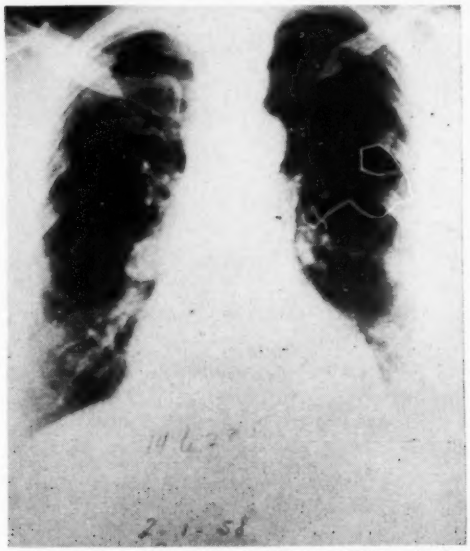

Figure 3.—(Case No. 4, M. B.) EPA of chest on admission, February 1, 1958.

pleural cavity. She was begun on anticoagulants, heparin and hedulin, and showed slight improvement. A few days later she developed pain in the right lower anterior chest, which was worse on deep breathing. X-ray film (Figure 4) revealed

Figure 4.—(Case No. 4, M. B.) Left pleural effusion present after first pulmonary infarct on left side, February 21, 1958.

fluid in the right pleural cavity. Two right thoracenteses were done with removal of 300 to 500 ml. of bloody fluid each time. Bilateral femoral vein ligation was done, and she became slightly more comfortable, but was still dyspneic. Because

of continuance of symptoms, an inferior vena cava ligation was done, and a striking improvement in dyspnea and tachypnea was noted. (Figures 5 and 6) A few days later she was ambulated, elastic stockings were applied, and at present she is in good health. Her total hospital stay was seven and a half weeks.

Figure 5.—(Case No. 4, M. B.) Bilateral pleural effusion, the left one subsiding, but a large amount of fluid is present in the right pleural cavity, March 3, 1958.

Figure 6.—(Case No. 4, M. B.) Right pleural effusion decreased in amount after two thoracenteses, March 18, 1958.

Comment: This patient was originally thought to be in congestive failure. The syncopal attacks and calf tenderness alerted the clinician to the possibility of phlebothrombosis and pulmonary embolism which was confirmed later by the physical findings of pulmonary infarction in the left lung with pleural fluid, then a few days later fluid on the right. Digitalization had no effect on her course. She did not improve appreciably on anticoagulants or bilateral superficial vein ligation or thoracenteses. Because of this an inferior vena cava ligation was done and her subsequent improvement was dramatic.

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IMPORTANCE TO PATIENT CARE OF A MEDICAL EDUCATION PROGRAM IN NONUNIVERSITY AFFILIATED HOSPITALS *

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The basic philosophy underlying any medical education program in a nonuniversity affiliated hospital is to provide for the House Staff continuing educational facilities which will adequately equip them to apply more intelligent care to the patients in the hospital. The demand for this scientific training by the House Staff is simply predicated on their need of this progressive knowledge to provide for the adequate care of the patients entrusted to them. The motivating force behind the efforts and interest of the Visiting Staff is their need for intelligent assistance and scientific service for their own individual patients whose very lives are their personal responsibility.

It is obvious that good patient care is entirely dependent upon the quality of these two important functions: namely, the demand and need of the Visiting Staff for medical service to their patients, and the demand and need of the House Staff for continuous, effective, adequate and desirable training. The latter provides the House Staff with the necessary proverbial tools to effectively perform their most important function of ably assisting the Visiting Staff in the many problems involved in rendering good patient care. These two seemingly diametrically opposed objectives are not opposing forces; but actually are supporting forces. Certainly the demand for service and the demand for education must be met, equally and continuously, for the sole reason that improvement in one necessarily improves the other. The thorough comprehension of these two basic facts, by all partners concerned in the providing of medical care to their patients is absolutely essential. The intelligent, cooperative effort of all proponents of any organized program will provide the necessary studies and ultimate solution to the problems of effective administration of a desirable medical education program, absolutely necessary to good patient care.

When one investigates the history of previous endeavors, both the success and failures, by nonuniversity affiliated hospitals to facilitate medical education programs, one conspicuous thread is evident throughout the fabric of their administration; and that is the purpose for which the program was instituted. When the purpose: improvement in patient care, was foremost in the minds of all participants, the program has usually been successful. When the purpose of improvement in patient care was lost sight of, and other factors motivated the program; namely, elements such as personal aggrandizement, pride, envy, and other deleterious emotional factors, utter failure resulted.

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MEDICAL EDUCATION VS. MEDICAL SERVICE

Dr. John C. Leonard,[1] Director of Medical Education at Hartford Hospital, Hartford Connecticut, makes the following

pertinent observations: "Any sound program for graduate training in the non-university hospital obviously must function effectively from several different viewpoints. (1) It must function effectively from the viewpoint of those who are receiving the training, else it will die a natural, even though painful death. (2) It must function effectively in constantly improving patient care, else its entire structure will prove to be a sterile one. (3) It must function effectively in its role of stimulation of the hospital's attending staff, or it will have fallen far short of its goal. (4) It must function effectively for the greater hospital and community medical good, or the hospital administration and its Board of Directors will not be able to countenance the added cost of the program to the hospital and its community."

As stated by Dr. William J. Lahey,[2] Director of Medical Education at St. Francis Hospital, Hartford, Connecticut: "The demand for interns is becoming greater and greater. The Federal Service and the Medical School Hospitals have relative success because of the improved caliber of training and salary, by the former; and the fact that graduates continue to feel that the latter offer the highest quality of training. These factors have excited the criticism that these hospitals are monopolizing graduates and that the latter are oversold on the 'prestige factor' and do not receive a well-rounded internship. The fact remains, however, that an atmosphere of education and investigation continues to be the most powerful attraction year after year."

Dr. Harold Jeghers,[3] Professor of Medicine at Georgetown University, has contributed a vitalizing treatise as follows: "One hears statements like this: 'Our primary concern is to the patient,', or, 'Medical education, medical research is all right, but our first duty is to the patient!' At first glance this seems like a wonderful thought to have! The point which is overlooked is that without current medical education, the patient receives 1940 care in 1958. There is just no way patients can get good care unless education goes on. What was 'good care' last year may not be good care this year. The basic premise is that those hospitals with good medical educational attitudes and facilities automatically secure better care for their patients. With continuous education, any physician practices better medicine.

"Essentially, hospitals exist to provide medical care and nursing service not possible nor practical at home nor by ambulatory care directed from a physician's office. Patients are unaware of the enormous ancillary activities which are needed to make this care possible." Regardless of who supplied the patient's care, it is ultimately the responsibility of the patient's physician. In the public mind, he is responsible; he is the source from which their healing comes; he must not fail his responsibility. His interest in medical education and scientific activity in the hospital is his only source of authority which is so necessary in complementing this responsibility.

HOUSE STAFF TRAINING AND GRADUATE STUDY

Of what value is a hospital medical education program to the members of the Visiting Staff of the hospital? Dr. Jeghers also gives an explicit answer to this question:

"Physicians commonly get caught in the whirl of an active practice and find it increasingly difficult to keep abreast. Yet one of the most certain things about the practice of medicine is that its basic concepts are continually changing. One must move along with these changes. To remain stationary, is to fall behind. There is a great need in this country for the development of mechanisms whereby a physician can do continuous post-graduate work throughout his professional career."

The following is a paraphrase of the three points Dr. Jeghers proposes as the means by which the practicing physician can obtain post-graduate medical education:

1. Reading gives one a fair concept of new things, but no intimate, practical de-

tails nor the skill to perform any intricate procedures.

2. Post-graduate courses are expensive in time and money. After days of lectures, one's grasp of the subject is limited by cumulative fatigue.

3. Post-graduate daily study and teaching in one's own hospital staff activities, with his colleagues and friends in medical education programs from year to year is most inviting and desirable.

The advantages of this third avenue of study are obvious to all of us. Medicine in its broadest sense can be broken down into three components: The first is finding new facts — medical research. This may be basic science research, clinical research, or it might be the everyday research doctors do constantly by evaluating something in terms of their own experience. The third component is the practice of medicine. The reason for finding these facts is to apply them to the care of the patient throughout the practice of medicine. It is almost impossible, however, for the busy practitioner to sort out the practical procedures he needs from the highly technical basic scientific research and apply them to his patients. Therefore, the second component (which links medical research to medical practice) is the intermediary role played by medical education. This intermediary role, played by medical education, is the one place wherein all three phases may be joined daily in study and teaching, each contributing a part of its own intellect and experience to the other's problems, and the solution thereof. Actually, its function is to transmit basic information to the research students, graduate students and practitioners of medicine in an understandable fashion.

Certainly the aforementioned foundation stones of a Medical Education Program must be provided in order to acquire an adequate superstructure, but, unless the adequate superstructure is carefully planned, the foundation stones alone will not of themselves create a practical and serviceable edifice.

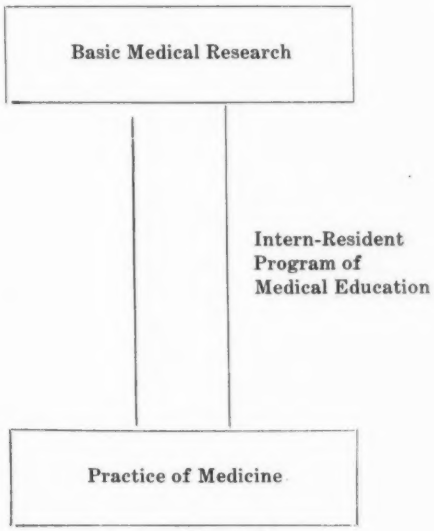

Figure 1

HOUSE STAFF POSITIONS AND AVAILABLE PERSONNEL

One criticism pointed out by Burgess and Leonard,[4, 5] which has been repeatedly made against many well equipped nonuniversity affiliated or community hospitals is that these institutions heretofore have employed physicians from both United States and foreign medical schools, as interns and residents, exploit them for their services alone, and give them no real training in return. Furthermore, many of these hospitals do not even have any instruction or formal supervision for the interns and residents that they do obtain. That this criticism is still true, in some areas, there is little doubt. Much needs to be done in these areas to solve the existing problems which excite this criticism.

Realizing the attraction that medical education[6] exerts in obtaining sufficient house staff personnel to provide adequate patient care, it is indeed gratifying to see hospital administrators and the attending medical staffs cooperating and coordinating their efforts in time and money to build attractive and desirable medical education programs in their respective hospitals. The majority of the nonuniversity affiliated or community hospitals have come to realize that unless they pro-

vide a desirable medical education program for their interns, their chances of obtaining interns in the future are very faint indeed. They well understand that a vital progressive educational program for interns provides an excellent preparation for residency training in the same hospital or a parent accredited institution. Furthermore, it is a generally accepted fact by all partners interested in medical education, that there in one individual, and only one individual, who can attract prospective interns to apply for internship in a given hospital. That individual is the intern who is fully satisfied with the training he is receiving or has received. His satisfaction and his recommendations are directly proportional to the character and quality of the training program administered by the hospital.[7]

The fact remains, however, that many well equipped nonuniversity affiliated or community hospitals with desirable and attractive medical education programs are unable to obtain House Staff personnel of the caliber and in the numbers required for good patient care.

Burgess and Leonard[4, 5] point out that in the New England states and in general, throughout the rest of the United States, the nonuniversity affiliated hospitals (more properly termed community hospitals) take care of four to five times more patients in a given community than university affiliated hospitals. It seems paradoxical to them that nonuniversity affiliated hospitals have decreasing numbers of American medical school graduates as interns and residents, while each year finds increasing numbers of medical school graduates remaining under the maternal wing of the university hospital of their own or another medical school. They also state that it has been admitted by some medical school faculty members who are also chiefs of their respective services in university hospitals, that they have increased their quota of interns, so that if they fail to secure a maximum number, they may continue unhampered because they still secure an adequate number of interns. Those who spend a considerable amount of time in university as well as nonuniversity hospitals, feel that most university hospitals are overstaffed and most nonuniversity hospitals are understaffed.

However, here in Louisiana, the trend appears to be in still another direction. We find that between 40 and 60 per cent of medical school graduates do not remain with their parent university affiliated hospital. The hospitals of the United States Army, the United States Navy, the United States Public Health Service, Federal Security Agency and Veteran's Administration are becoming increasingly more attractive to American medical graduates. It is becoming increasingly apparent that the majority of the graduates are already married at the time of graduation or very soon thereafter. In most instances, their family responsibilities demand that salary, in addition to training, be an important consideration. Notwithstanding this fact, we know that salary should not be the motivating factor, at the sacrifice of his training which he also sorely needs.

FOREIGN MEDICAL SCHOOL GRADUATES

Physicians who are graduates from foreign medical schools, have been employed extensively by hospitals throughout the United States to help fill the need for House Staff personnel.[8] Some of the foreign interns presently accepted will no longer be available to us because of the realistic ruling by the Council of Medical Education and Hospitals of the A.M.A. disapproving prospective foreign interns, until they pass a satisfactory language examination, in addition to qualifying tests as to their medical education and medical background.

A recent communication from the executive secretary of the National Intern Matching Program, Inc.,[9] states the "New Policy Regarding Foreign Student Participation" as of May 2, 1958, to be as follows:

"Graduates of foreign medical schools wishing to enroll in the 1959-60 matching program must be certified by the Educational Council for For-

eign Medical Graduates. Hospitals enrolled in the National Intern Matching Program are **not to offer** internship appointments to non-participating foreign medical school graduates until after the matching program results are announced on March 16, 1959."

This screening procedure, when fully implemented, will insure a source of the better qualified applicants for training. Many nonuniversity hospitals are now endeavoring to acquaint the top English speaking foreign graduates with their training facilities. The foreign schools are now in the process of improving their educational facilities in order to improve their standards. They would desire their top graduates to study in the hospitals with well organized training programs in the United States, and, upon completion of their training, to return to their parent institutions as teachers.

INTERN TRAINING IN COMMUNITY HOSPITALS AND RESIDENT TRAINING IN UNIVERSITY HOSPITALS

One question which has excited considerable interest over the years is as follows: What other studies are in progress to help the nonuniversity affiliated hospitals with their problems involved in obtaining adequate House Staff personnel from United States medical schools to administer good patient care?

The House of Delegates of the Louisiana State Medical Society, at the request of the members of the House of Delegates from the Orleans Parish Medical Society, is now studying this problem in cooperation with medical schools and other interested bodies both on a local and national level. A brief of the proposition submitted for study may be best understood if it were recorded here as originally presented:

SENIOR MEDICAL STUDENTS IN "CLINICAL CLERKSHIPS"

"One of the most important problems facing the administrators and Medical Staffs of the nonuniversity connected hospitals in the State of Louisiana, is their inability to attract interns and residents of the caliber and in the numbers needed, to provide the essential service in a carefully planned and well organized House Staff training program in keeping with good patient care in their respective hospitals. This is not an unpleasant temporary situation, but one that grows increasingly more serious from year to year in hospitals throughout the United States. The magnitude of this problem is well emphasized by the 1953 report of the Council on Medical Education and Hospitals of the A.M.A.[10] This report emphasizes the fact that in 1940, there were approximately 12,000 interns and residents on duty and very few, if any, internship and residency positions unfilled. Conversely, today, more than 25 per cent of all House Staff positions are unfilled. An even greater number of House Staff positions in nonuniversity connected hospitals remain unfilled. This situation exists in spite of the fact that the available number of interns and residents on duty has risen from 12,000 to 24,000 since 1940. Obviously, it is impossible for these 24,000 to fill the approximately 32,000 to 36,000 positions now open for House Staff service. Many factors influence this picture, but the principal cause producing this shortage of interns and residents is the longer period of training that has become necessary to meet the added demands of medicine, surgery and their respective specialties.

"Considerable literature and research have accumulated in the last few years on this subject. Special emphasis has been placed on the solution to this problem. Organized medicine has rendered considerable aid in suggestions for improving medical education programs in nonuniversity connected hospitals to make their hospital a more desirable place for intern and resident training. However, the challenge to these nonuniversity affiliated hospitals to meet the competition presented by the university connected hospitals would require a great outlay of money for a Medical Education faculty and other facilities to match those provided by a university connected hospital. An excellent training program, conducted by the Visiting Staff for interns, can be easily supplied in most nonuniversity modern hospitals. This type of training

program must be supervised by an individual especially trained in Medical Education.

"Exciting the most interest at this time among the medical profession [11] is the proposal that the university connected hospitals avail themselves of the proposition through which the medical students from their affiliated university would act as "clinical clerks" in the hospital and actually do the work which heretofore has been done by interns. They are designated as "clinical clerks" and reside in the hospital as the present interns do, rotating by means of the "block system" or any appropriate rotation system as presently used for interns. This will make it possible for the medical schools to afford added practical experience for their senior students; and, furthermore, for the university connected hospitals to place emphasis on their residency educational programs. The nonuniversity affiliated hospitals would then place emphasis on their intern medical education training programs. The latter group will receive excellent intern training in the nonuniversity associated hospitals which can easily facilitate an excellent intern educational program. Under the supervision and training in a well organized intern training program conducted by the hospital Visiting Staff, the excellent quality of the intern training will put them in a desirable position to compete for subsequent residency training in the university affiliated hospitals.

Two important facts immediately become evident:

1. The university connected hospitals would save considerable money in salaries to interns.

2. The medical schools will necessarily have to operate on a year round basis, using the "block" or other systems for vacation periods, etc., etc.

"It has been emphasized repeatedly that this proposal has been considered for many years and is not, at this time, too revolutionary to receive proper study. It is perfectly possible that this adjustment can be made gradually, so that all pitfalls and radical changes and modifications be carefully considered during the process of evolution in medical education.

"It is a general feeling that, should this proposition receive the proper study, investigation and consideration by all partners concerned in medical education, a reasonable and practical solution will result."

Already some of the many administrative bodies are actually engaged in this study. Certainly the above solution may not be final at all. Especially difficult would be the task that the State Boards of Medical Licensure would have to integrate such a program within the framework of the Medical Practice Act of their respective states. However, this solution, if desired, can be more easily implemented wherein the parent university completely controls, as if by ownership, the affiliated hospital in which their students are taught.

Since we all agree that all sick people need and should have good patient care, it follows that the one single objective of all partners in medical education is ultimately the building of facilities needed in healing of the sick individual. When all the individuals and groups of administrative bodies and medical schools attack the problem in a dynamic and cooperative manner, a sound and desirable solution is usually forthcoming.

SUMMARY

1. One of the major problems facing the administrators and attending staff members of nonuniversity affiliated hospitals (more properly referred to as community hospitals) is their inability to obtain house staff personnel of the caliber and in the numbers needed to render the service necessary to good patient care.

2. An atmosphere of medical education and clinical investigation in the nonuniversity hospitals continues to be the most powerful attraction to prospective house staff personnel from year to year. However, we must continue to recognize the attractions of material assets, such as

salary, living quarters, food, etc.

3. The value of a well organized, carefully planned, and ably executed medical education program to both house staff personnel training and attending staff post-graduate daily study is emphasized.

4. Thusly, the demands for service and the demands for education and clinical investigation must be equally satisfied if good patient care is to be achieved. In fact, the providing of educational and clinical investigation programs demands paramount consideration.

5. The number of graduates of foreign medical schools available for house staff service will be limited by a recent ruling of the National Intern Matching Program, Inc.

6. Organized medicine, on the local and national levels, in cooperation with medical schools and their affiliated hospitals, should continue its efforts with the administrative bodies of medical education and hospitals in the study of this house staff problem and its ultimate solution.

7. One such study of the use of senior medical students as "clinical clerks" in respective university affiliated hospitals is in progress.

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DIURIL*—A NEW ANTIHYPERTENSIVE DRUG

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This study consists of observations on 22 cases of essential hypertension. The majority of these patients were seen in the Out Patient Hypertension Clinic at Charity Hospital and the remaining ones in our own private practice. Negro and white patients of both sexes comprised our study group. These patients were observed at weekly intervals for a period averaging 4.2 months. All of these patients had been followed for several months to several years prior to their selection for the study. The blood pressure levels and the normal variations of these levels in individual patients were, of course, known to us. This group fulfilled the criteria for essential hypertension, and the evidence for irreversible renal and vascular damage was lacking in all cases. Diuril alone was used in this study, some cases receiving as much as 1500 mgms. daily for several weeks.

A breakdown of our cases may be seen in Table 1.

TABLE 1
TOTAL 22 CASES

Race and Sex	W.M.	W.F.	C.M.	C.F.
No. of cases	2	5	5	10
Average age	37	46	32	43
Average B.P. pre Diuril	195/103	154/112	185/125	215/146
Average dosage mgm/24 hr.	750	750	1400	1225
Average duration of therapy in months	3½	5½	3	4

Hypertension Clinic, Louisiana State University Medical School Unit of Charity Hospital of Louisiana at New Orleans.

*Merck Sharp & Dohme.

In 3 cases an initial response varying from 20 to 40 mm.Hg. systolic and from 10 to 40 mm.Hg. diastolic was seen. One of the 3 cases failed to maintain a response; another had a slight sustained reduction of blood pressure levels of 20 mm.Hg. systolic and 40 mm.Hg. diastolic; the other had a sustained reduction of 60 mm.Hg. systolic and 10 mm.Hg. diastolic.

Our observations on the use of Diuril as a hypotensive agent have led us to the conclusion that there is no consistency in its hypotensive effect. In the vast majority of instances no appreciable blood pressure lowering was noted. Where there is some diminution in blood pressure levels, it has been our impression that there is an associated diuretic effect in a patient with fluid and sodium retention.

Diuril proved to be an excellent diuretic in our experience in patients with fluid retention, not only in this series but in other cases in whom we have used the drug for its diuretic effect. In this series no toxic effects from Diuril were noted.

DIABETES AND PREGNANCY *

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New Orleans

INTRODUCTION

The purpose of this paper is to present the results of our recent experiences at Charity Hospital in New Orleans in the management of the diabetic who becomes pregnant, and to re-emphasize some of the special problems encountered in her care. It is well known that the fetal mortality and complications seen with diabetes increase with the severity of the disease. Therefore, in order to compare experiences with other clinics, some form of classification of the severity of diabetes is necessary.

White's classification (Table 1) is, by and large, the most suitable at this time, although it does not take into account the amount of insulin required, or the ease or difficulty with which control is maintained. Both of these factors, I feel, are important not only in grading the severity of the disease, but also as an aid in formulating some sort of prognosis in the gravid diabetic. This report concerns 50 consecutive diabetics admitted to the labor room, and covers a period from July 1956, through February 1958. Table 2 shows the diabetic severity and fetal mortality of the group. The number of cases in Classes A and D are so small as not to be significant. Ten to fifteen years ago, pregnancy complicated by diabetes was a fairly rare condition. With improved care from the obstetrical point of view, and better medical management as well as pediatric care in the neonatal period, more and more women with diabetes are able to conceive and successfully have families. This is bound to be reflected through the years by an increased number of women who have latent as well as overt diabetes in the childbearing years. This was graphically brought out by Adams [4] who reported that in one hospital

TABLE 1.
CLASSIFICATION OF DIABETIC PREGNANCY

Class	Severity
A	Requiring no insulin and little dietary regulation
B	Onset after age 20; duration less than 10 years
C	Onset between ages 10 and 19; duration between 10 and 19 years
D	Onset before age 10; duration 20 years or more
E	X-rays demonstrate calcification of pelvic arteries
F	Complicated by nephritis

TABLE 2.
FETAL MORTALITY AND DIABETIC SEVERITY

Class	No. cases	Fetal deaths	
		No.	%
A	2	1	50
B	34	9	26.5
C	12	6	50
D	2	1	50

* Presented at the Seventy-eighth Annual Meeting of the Louisiana State Medical Society, in Shreveport, May 6, 1958.

in a ten year period from 1941-51, only 50 diabetics reached twenty-eight weeks' gestation or more. In the next year, however, there were 22, and in the next two years an additional 29. Thus, in a period of three years, more diabetics reached at least twenty-eight weeks of pregnancy than there had been during the previous ten years.

COMPLICATIONS

FIRST TRIMESTER

Diabetes may become manifest at any time during pregnancy. At each visit, it is mandatory that the urine specimen be tested not only for albumin but sugar as well. In the present series, there were 18 women diagnosed for the first time as diabetic during this pregnancy. All 18 were picked up because of glycosuria sometime during their pregnancy, and the diagnosis confirmed by glucose tolerance tests. The earlier diagnosis is made and the sooner the patient presents herself for care, the better the chances will be for a successful outcome. Pedersen [5] has shown that fetal survival is three times better when the diabetic is seen relatively soon after she becomes pregnant. In his series, patients who were seen early in pregnancy had an 11 per cent fetal mortality. Those who came in for their initial visit later had a fetal mortality of 36 per cent.

Abortion. It was thought for many years that patients with diabetes were more apt to have early abortion. Some authors today, and most textbooks, state that early fetal wastage is higher in the diabetic than the nondiabetic group. The 50 women in this study had 267 conceptions with only 34 abortions, a rate of 12.7 per cent. This is no greater than that experienced in the population at large, so that I would agree with those who maintain that the abortion rate seems to be no higher in the diabetic than in uncomplicated pregnancies.[3-6]

Carbohydrate Tolerance. During the first trimester, a change in carbohydrate metabolism is to be expected. Usually sugar tolerance decreases causing a need for more insulin, but rarely the converse may be true. Many diabetics become labile during pregnancy, and require constant watching to prevent acidosis on the one hand and insulin shock on the other. Should nausea and vomiting occur during this time, the patient must be watched very carefully. Ketosis and acidosis are extremely hazardous during any phase of gestation. The leading cause of maternal death in diabetes is still acidosis. It is mandatory that these patients be hospitalized and regulated promptly.

SECOND TRIMESTER

It is during this period that the basal metabolic rate begins to increase, and there may be a need for more carbohydrate in the diet. At about the fourth or fifth month, the renal threshold for sugar begins to decrease. To keep the urine free of sugar at all times is not only difficult, but such rigid management often causes the patient to become restless, tense, and discouraged. If allowed to spill a little sugar occasionally, she feels better and is happier. This implies that a little leniency in management is indicated, but by no means should all controls be cast aside. It is in the second trimester that the carbon dioxide combining power of the blood decreases, making acidosis more likely. The patient should be seen every week so that any changes in sugar tolerance may be discovered early. Because of the lowered renal threshold for sugar, and the decreased carbon dioxide combining power of the blood, acidosis may develop much more rapidly than in the diabetic who is not pregnant.

THIRD TRIMESTER

The third trimester is usually associated with the most difficult complications and decisions. We know that, for some reason, the blood vascular system in the diabetic seems to age more rapidly than that of the nondiabetic. Just as surely, it also follows that the placenta in most diabetics seems to age prematurely. Many of these placentas are large and have both old and fresh infarcts as well as scattered deposits of calcification. None of these changes are specific for diabetes; however, they seem to occur earlier in women with this disease. Although many detailed studies in histopathology and physiology of the pla-

centa have been carried out, we still know next to nothing concerning these fundamental changes. It is possible that this premature aging can explain many of the late complications of diabetes such as the tendency to develop toxemia, hydramnios, and death in utero.

Polyhydramnios. Polyhydramnios is noted more frequently in the diabetic pregnancy. There was a clinically significant increase in amniotic fluid above normal in thirteen of our patients (26 per cent). This is higher than the usual estimate of 15-18 per cent. One patient was successfully tapped six times because of extreme distention, and went on to deliver uneventfully two weeks later. Another patient that had to be tapped once at thirty-two weeks went into spontaneous labor, and delivered a 5 lb. 3 oz. infant which was stillborn.

Toxemia. There is a definite tendency for the diabetic to develop toxemia. Our usual incidence at Charity Hospital is around 18 per cent. The incidence in this series was 38 per cent, or double that usually seen. This seems to be true over most of the country; however, the incidence of toxemia in a private series and in the north is much less. Why women with diabetes have a tendency to develop toxemia more frequently has never been answered. Perhaps it is related in some way to the concept of premature aging of the placenta.

Large Baby. It is a well known fact that diabetic and prediabetic women tend to have large babies. The cause of this phenomenon is still obscure, but does not seem to be related to hyperglycemia. Many prediabetic women have large babies with no evidence of hyperglycemia during pregnancies which antedated the development of diabetes. Snyder [8] and Hoopes [9] have shown that injections of pituitary gonadotropes in animals cause an increase in fetal weight at term. It seems evident that diabetes is a disease which not only involves the pancreas, but also is associated with a general endocrinopathy in which many glands of the body are involved, particularly the pituitary and the adrenals. These fetal giants are fragile and do not tolerate even minor degrees of trauma during labor and delivery. Difficult delivery of a large fetus through the birth canal is still one of the major causes of fetal loss. In this series, there were 18 babies weighing over 4,000 grams, with a loss of 8 or 44.4 per cent. There were 5 cases of shoulder dystocia with 3 intrapartum deaths, 1 Erb's paralysis, and 1 apparently unaffected infant. It seems probable that cesarean section would have circumvented the 3 intrapartum losses and prevented the brachial plexus palsy.

The radiographic demonstration of the presence of the distal femoral epiphysis, as pointed out by Adams,[4] eliminates much of the danger of prematurity. The real problem is that of the oversize infant which often is not recognized, and therefore not treated as it should be. It still is difficult to estimate accurately fetal size in utero. The presence of a large baby is often suspected, but the frequent association of polyhydramnios makes clinical evaluation of fetal weight no more than a guess. The need for more accurate methods with which to estimate fetal size is a very great one, and traumatic deliveries might thus be prevented. It is imperative that all of us who do obstetrics be thoroughly acquainted with the early recognition of shoulder dystocia and the methods available for overcoming this most serious complication.

Death in Utero. The risk of sudden unexplained death in utero is a very real hazard after the beginning of the thirty-eighth week of gestation. Again, the etiology of this occurrence is probably best explained by the concept of premature aging of the placenta. Planned early delivery from the thirty-fifth through the thirty-seventh week has greatly improved fetal outlook. Even so, many patients are still allowed to go beyond thirty-eight weeks, or even longer. In this series, there were 7 spontaneous deaths in utero in 25 patients who went to thirty-eight weeks and beyond, or an incidence of 28 per cent. To be faced with the decision of early delivery accompanied by the chances of prematurity, or risk the ever present threat of sudden death in utero, at times taxes the judgment of the obstetrician to the utmost.

Management. It is our policy, whenever possible, to admit these women at thirty-five weeks gestation. Up to this time, they have been followed in the out-patient clinic for special obstetrical problems. Our patients represent the indigent of Louisiana and, unfortunately, many of them do not seek help until very late in pregnancy. Their diets and insulin requirements are carefully regulated. Any anemia present is corrected, and the patient is gotten into the best possible condition before delivery. If, after stabilization in the hospital at thirty-five weeks, there is (1) no evidence of toxemia, (2) if the diabetes is under control, (3) if there is no evidence of hydramnios, (4) and if the baby is not excessively large, they are followed another week. Close watching and frequent evaluations are of the utmost importance during this time. If at any time from the thirty-fifth week through the thirty-seventh week, the diabetes gets out of control, the patient develops toxemia or hydramnios, or the infant becomes excessively large, delivery should be carried out. If all these criteria remain favorable, the patient is watched to the thirty-eighth week, at which time pregnancy is terminated.

Delivery. Approximately twenty-four to forty-eight hours prior to delivery, the patient is placed on divided doses of regular insulin in preparation for labor or cesarean section where dietary intake is limited. A sterile vaginal examination is done, and if the cervix is ripe, the pelvis adequate, and the head engaged, the membranes are stripped and ruptured, slowly draining off as much fluid as possible. Pitocin drip is given with the usual precautions. If, on examination, the cervix is not ripe and the patient not easily inducible, a cesarean section should be done. The section rate in these 50 cases was 14 per cent, which is probably too low.

After delivery, regular insulin is continued until requirements again become stable. Fully 75 to 80 per cent of patients improve their sugar tolerance in the immediate puerperium. The usual antepartal dose of long acting insulin will cause frequent episodes of shock. For this reason, frequent smaller doses of regular insulin are continued until carbohydrate metabolism again becomes stable.

Newborn. The newborn infant must be watched very carefully, especially during the first three days of life. These babies, many of whom are large and healthy appearing, have a tendency to behave much like prematures. They are sluggish and tend to be lethargic, and thus prone to develop anoxia. The rate of hyaline membrane disease is increased. They should be kept in a humidity controlled incubator, and stimulated often to cry and breath deeply. Hypoglycemia is no longer felt to play a part in the neonatal death of these babies. They should, however, have all the specialized care and attention of the most delicate premature infant.

Hormones. Any mention of the use of female sex hormone preparations has been omitted since none of these patients were given such therapy. There is still a great difference of opinion over the country as to the benefit derived from their use. Most obstetricians feel that endocrine therapy is not necessary. However, Priscilla White has presented evidence that they may be of value. In her earlier series without their use, the fetal mortality was 11 per cent.[1] In her later work in which hormones were used, the fetal mortality was 10 per cent.[2] In all fairness, it must be pointed out that the first series was done before her classification of the severity of diabetes, thus the two groups may not be comparable as far as having the same numbers of severe diabetics. If she could run another comparable series, giving the same meticulous individual care which is possible in her clinic, but without the use of hormones, then the problem would probably be solved once and for all.

CONCLUSIONS

The experiences at Charity Hospital in New Orleans over a twenty month period in the management of 50 consecutive pregnant diabetics have been presented.

The fetal mortality was 34 per cent with no maternal deaths.

The complications in each trimester, and variations in physiology have been dis-

cussed. It is felt that improved fetal salvage does not depend on any one technique or idea, such as the use of hormones or routine section at thirty-seven weeks. Best results are obtained by exercising meticulous care starting early in the antepartal period, coupled with careful medical and obstetric judgment through labor and delivery to be followed by detailed pediatric care through the neonatal period.

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EVALUATION OF GLUCOSE OXIDASE IN URINALYSIS

The qualitative determination of the presence or absence of glucose in the urine has long been a necessary part of every urinalysis. The methods in clinical use have been those of copper reduction, such as Benedict's and Clinitest. Under certain circumstances other substances appear in the urine which reduce copper, with the result that some uncertainty develops as to the interpretation. In many instances, the physician has come to regard a reaction recognized as a trace as probably being without clinical importance. Such findings and deductions render difficult satisfactory screening of patients in a systematic attempt to determine whether glycosuria exists or not.

In recent years the importance of diabetes has increased greatly in proportion to other diseases. This is true not only from the point of view of establishing a satisfactory treatment regime at the earliest possible time, but also, for the necessity of recognizing it in some of its various hidden forms. Consequently, a method of testing glycosuria which is specific for glucose is of great value.

Filling this need are two preparations commercially available, utilizing glucose oxidase, in urinalysis for qualitative determinations. These are available under the names of Tes-Tape and Clinistix. They have been in use for a sufficient time to be able to judge their value in comparison with the several methods previously available.

Reports by several investigators show that the glucose oxidase technique is specific for glucose, qualitatively accurate, and simple to perform. Several reports also show that the copper reduction methods, of which Benedict and Cinitest are examples, are less sensitive than the oxidase. Chertack [1] reported on over 1000 random specimens of urine collected from more than 500 patients in all trimesters of pregnancy, which were tested by the Clinitest and the glucose oxidase methods. Specimens which gave a positive reaction with any test were further studied by Benedict's, yeast fermentation test, osazone, Seliwanoff's test for fructose, Tauber's test for pentose, Escherichia coli treatment for lactose, and paper chromatography. With this accurate determination of the presence or absence of glucose in the urine, it was found that 196 urine specimens contained a reducing substance. Of these, in 135 samples that substance was glucose. From among the 135 proven to contain glucose, 134 were positive by the Clinistix method; 127 by Tes-Tape; and 30 by Clinitest; and 113 by the Benedict test. The remainder of the 196 specimens that contained some reducing substance, that is, 61, which did

not contain glucose also gave a positive reaction with the Benedict test. Accordingly, approximately one-third of the specimens of urine reacting positively with Benedict's did not contain glucose.

The sensitiveness of the oxidase method can be judged by the observation of Chertack that positive reactions occur in concentrations of glucose ranging from 0.01 per cent to 0.1 per cent, depending upon the amount of inhibitory substances present. This is to be compared with reactions which are positive between 0.15 per cent and 0.2 per cent, respectively, obtained with Benedict's and Clinitest methods. It is apparent that the glucose oxidase method is more sensitive and more specific for the detection of urinary glucose than are the copper reduction techniques. Their use, however, in quantitative determinations has not been reported as satisfactory because in some instances urines containing as much as 2 and 3 per cent glucose gave the same intensity of reaction as that obtained with 0.5 per cent glucose.

The clinician, therefore, has available a test which is specific and sensitive. When a positive reaction has been obtained the need exists to evaluate each patient individually and determine the significance of glycosuria in that case. Glucose oxidase tests are a valid screening method for the detection of urinary glucose and as such should be beneficially utilized by the profession.

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DIABETES DETECTION WEEK

The second week of November will be, as for some years past, Diabetes Detection Week. This is a commendable undertaking. It is sponsored each year by the American Diabetes Association, a national organization devoted to the problems of diabetes, in which the membership is limited to physicians. Intensive study of the problems of diabetes is also a part of its mission.

There is reason to believe that there are just as many diabetics undetected as there are now under treatment. The aim of Diabetes Detection Week is to supply the necessary stimulus to physician and patient to carry through a urinalysis, and when necessary, other diagnostic procedures.

This enterprise deserves the enthusiastic cooperation of all physicians.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

SECOND SESSION, 85th CONGRESS

The 85th Congress wrote an impressive number of health measures, the more important ones being listed below. It failed to act on some bills, and in all likelihood, these will be introduced anew in the 86th Congress convening next January 7.

SUBJECT	BILL NO.	HOUSE	SENATE
Public Works Loans	S. 3497	Voted Down 8/1	Passed 4/16
Civilian Pay (VA Doctors)	S. 734	Public Law 85-462, June 20	
Military Pay	H. R. 11470	Public Law 85-422, May 20	
Pub. Health School Grants	H. R. 11414	Public Law 85-544, July 22	
HEW Appropriations	H. R. 11645	Public Law 85-580, Aug. 1	
Union Health Plans	S. 2888	Public Law 85-836, Aug. 28	
Social Security	H. R. 13549*	Public Law 85-840, Aug. 28	

SUBJECT	BILL NO.	HOUSE	SENATE
Med. School Aid	H. R. 6874 S. 1917	Hearings Held	
Research Facilities	H. R. 12876	Public Law 85-777, Aug. 27	
Chemical Additives	H. R. 13254	Awaiting Presidential Signature	
Jenkins-Keogh Taxes	H. R. 10 S. 3194	Passed 7/28	
Hill-Burton Extension	H. R. 12628	Public Law 85-664, Aug. 14	
Hill-Burton Loans	H. R. 12694	Public Law 85-589, Aug. 1	
Federal Aviation Agency	S. 3880	Public Law 85-726, Aug. 23	
Civil Defense Aid	H. R. 7576	Public Law 85-606, Aug. 8	
Defense Reorganization	H. R. 12541	Public Law 85-599, Aug. 6	
Medicare Appropriations	H. R. 12738	Public Law 85-724, Aug. 22	
Nursing Home Loans	S. 4035 H. R. 13776	Voted Down 8/18 Pending	Passed 7/11
Presumption of Service Connection	H. R. 413 H. R. 1143	Passed 7/7 Passed 7/21	Postponed
VA Hospitalization	H. R. 10028	Reported 7/30	
Aging Conference	H. R. 9822	Signed Sept 2, Awaiting P. L. No.	

NO ACTION: * Medical Care for Aged (Forand's H. R. 9467); Grants and Scholarships for Nursing (H. R. 306); National Compulsory Health Insurance (H. R. 3764); Health Insurance Pooling (H. R. 6506 and H. R. 6507); Rehabilitation (H. R. 10608 and S. 3551).

Social Security Amendments

As it has done every election year since 1950, Congress again amended the Social Security Act. It increased Old Age, Survivors and Disability Insurance benefits by 7%, in response to demands that benefits keep pace with the cost of living. It provided an additional $197 million for public assistance recipients, and gave states greater flexibility in use of federal funds for financing the medical care of the aged, blind, the disabled and dependent children. To finance the liberalizations, the law increases the taxable base from $4200 to $4800 of gross employment earnings and raises the tax in 1959 from 2¼% to 2½% for the employer and employee, and from 3⅜% to 3¾% for the self-employed. Additional increases are scheduled for 1960, 1963, 1966 and 1969. By 1969, the self-employed will be paying 6¾% of earnings.

Congress left the door ajar for consideration by the next Congress of bills using the social security system to provide hospital and medical care for OASDI beneficiaries. An articulate minority wanted this enacted this year via the proposal of Rep. Aime Forand (D., R. I.), but Congress decided against it. However, the House Ways and Means Committee directed the administration to make a study and report by next February on the various possibilities for financing medical care for the aged, with particular emphasis on the possible practicability of increasing OASDI taxes and using the money to purchase health insurance on retirement. The AMA took a strong stand against using the social security system to provide such care, viewing it as a beginning of national compulsory health insurance.

Medical School Construction Aid

Bills to authorize one-time grants for new and existing medical schools to build classrooms were pending in both House and Senate at adjournment. Extensive hearings were held by the House Interstate health subcommittee. No bill was reported to the floor because of (1) concern over the segregation issue, (2) a question of whether the states have exhausted all resources, and (3) a reluctance of some committee members to spend the money now. The AMA supported one-time grants in its testimony.

Jenkins-Keogh Tax Deferral

The drive to give the self-employed equal treatment with the employed in setting aside tax-deferred sums for retirement plans moved closer than ever to enactment. A bill passed the House late in July with only a smattering of opposition. But it died in the Senate Finance Committee; a major factor was Treasury Department insistence it would result in a large tax revenue loss. AMA, in concert with the American Thrift Assembly, pressed hard for enactment. New efforts to get the measure passed in the 86th Congress are being planned.

Medicare Appropriations

Congress took a long look at the medicare program, now nearing its second anniversary, and decided that the civilian phase of the program had to be curtailed in favor of greater use of military facilities. The House first trimmed $10.2 million from an admittedly slim budget of $70.2 million,

and then wrote into the Defense Department appropriation a section that would have prevented the services from asking for any supplemental appropriation for the entire fiscal year or using other funds. When it reached the Senate, Medicare and Defense officials joined with the American Medical Association and others in a plea for restoration of funds and elimination of the destructive section. This was accomplished through an amendment sponsored by Senator Knowland (R., Calif.). In the ensuing conference, language was written in a report which, while not having the full force of the law, nonetheless directed the military to start cutting back on some aspects of civilian medicare. To this end, the Office for Dependents Medical Care announced new restrictions effective this fall. They aimed at higher military facilities utilization, such as requiring dependents living with sponsors to use military resources unless they are not available.

Veterans Hospitalization

Late in the session as a windup to lengthy hearings on veterans entitlement to VA hospitalization, the House Veterans Affairs Committee reported out an omnibus hospitalization bill. The measure had as its major objective the opening of some 5,000 additional beds which the committee maintains the Budget Bureau is holding back on. It was reported too late for final action. The AMA testified in favor of a clear spelling out by Congress of veterans entitlement to federal care.

White House Conference on Aging

The President is instructed to call a White House Conference on Aging in January, 1961, by a measure passed late in the session. This national meeting will bring together federal, state and local leaders working in the field of aging. Their objective: to arrive at facts and recommendations on the utilization of skills, experiences and energies, and the improvement of the conditions of older people. A final report would be submitted to the President within 90 days after the conference. A series of state-organized meetings would precede the 1961 conference. To help the states finance these meetings, the law provides up to $15,000 a state with a minimum of $5,000. These figures, reduced from the original $50,000 per state, were used with the anticipation that states would also participate substantially in the financing. The AMA gave this legislation its full support; author of the bill was Rep. John Fogarty (D., R. I.).

Doctor Draft Extension

Under this act passed in the first session, selective service has authority until July 1, 1959, to call certain physicians up to age 35 for military service. Only those doctors with obligations under the regular draft and who have been deferred for any reason may be called. Although the military has been getting enough doctors through new graduates and volunteers, it expects to ask for another extension next year.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Ascension	Third Tuesday of every month	
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

CANCER SEMINAR
ACS Alabama Division
January 28-29, 1959

Plans have been worked out by the American Cancer Society, Alabama Division, and the Medical Association of the State of Alabama to hold a cancer seminar in Birmingham, January 28-29, 1959, headquarters at the Dinkler-Tutwiler Hotel. Outstanding speakers have been obtained for this event and are as follows:

Dr. Alan Thal, University of Minnesota School of Medicine

Dr. George C. Andrews, New York City

Dr. Oliver Moore, Memorial Hospital, New York

Dr. Thomas F. Nealon, Jr., Jefferson Medical College

Dr. Louis A. Leone, Medical College of Virginia

Dr. Milton Friedman, New York University School of Medicine

Dr. Herbert C. Schmitz, Loyola Medical School

Dr. Warren Cole, University of Illinois School of Medicine

Dr. William W. Scott, Brady Urological Institute, The Johns Hopkins Hospital

Complete details will be available at a later date from the office of the American Cancer Society, 2029 Warrior Road, Birmingham, Alabama.

GASTROENTEROLOGICAL CONVENTION

The 23rd Annual Convention of the American College of Gastroenterology will be held at the Jung Hotel in New Orleans, La., on October 20, 21, 22, 1958.

In addition to the many individual papers to be presented, there will be panel discussions on Gastric Carcinoma, Steroids in Gastroenterology and Functional Disturbances of the Gastrointestinal Tract. There will again be scientific as well as commercial exhibits and the sessions will be open to all physicians without charge.

On October 23, 24 and 25, immediately following the Convention, Dr. Owen H. Wangensteen of Minneapolis, Minn. and Dr. I. Snapper of Brooklyn, N. Y., will again be the moderators of the Annual Course in Postgraduate Gastroenterology. The sessions will be held at the Jung Hotel and in the Auditorium of the Louisiana State University School of Medicine. Attendance at the Course will be limited to those who have registered in advance.

Silver certificates are to be presented to those who have been affiliated with the organization for twenty-five years.

Honorary Fellowships are to be presented to Major-General James P. Cooney, M.C., Deputy Surgeon-General, Department of the Army, Washington, D. C.; Dr. James D. Rives, New Orleans, La. and Dr. Edgar Hull, New Orleans, La.

As a part of this year's sessions, a one-day regional meeting will be held at the University Hospital in Mexico City on October 27, 1958, and members of the College from that city will present papers.

Copies of the program and further information concerning the Postgraduate Course and Mexico Regional Meeting may be obtained by writing to: American College of Gastroenterology, 33 West 60th St., New York 23, N. Y.

TWELFTH ANNUAL ASSEMBLY LOUISIANA ACADEMY OF GENERAL PRACTICE

The Louisiana Academy of General Practice will hold its Twelfth Annual Assembly on October 21-23, 1958 at the Bentley Hotel in Alexandria, Louisiana. The Congress of Delegates will meet on the morning of October 21, and this meeting will be followed by a scientific assembly which will close on the afternoon of October 23.

Following are the speakers and their topics:

Don W. Chapman, M.D.—**Anxiety Heart Disease and Management of Myocardial Infarction**

Seymour Hershman, M.D.—**Hypnosis** and **Practical Demonstrations in Hypnosis**

Isadore Dyer, M.D.—**Emergencies in the Third Stage of Labor** and **Surgery in Pregnancy**

Ellard Yow, M.D.—**The Use of Steroids in Systemic Diseases** and **Recent Advances in Therapy**

E. Sinks McLarty, M.D.—**Postgraduate Training and the Preceptorship Program**

W. W. Frye, M.D. and M. E. Lapham, M.D.—**Discussion of Dr. McLarty's Paper**

Jose Garcia Oller, M.D.—**Neurological Examination** and **Common Brain Tumors**

F. Michael Smith, Jr., M.D.—**Anemia, Vomiting and Diarrhea in Infants and Intravenous Fluids** and **Pediatrics Potpourri**

State officers for 1958-1959 will be installed on Wednesday evening by Dr. Fount Richardson of Fayetteville, Arkansas, President-elect of the American Academy of General Practice. Dr. Francis M. Brian of Alexandria, Louisiana, is Convention Chairman and Dr. Esmond A. Fatter of New Orleans is Chairman of the Scientific Assembly.

The scientific program has been accepted for fourteen (14) hours Category I credit by the Commission on Education of the American Academy of General Practice.

IMPORTANT NOTICE

The Executive Committee of the East Baton Rouge Parish Medical Society requests that the doctors of the state, their families and friends vote against the amendment providing for a Charity Hospital in Baton Rouge, in the November election, for the following reasons:

1—This is a ¾ mill state property tax.

2—The percapita tax burden in Louisiana already far exceeds that of most other states. We are spending approximately 30 million dollars to support existing charity hospitals which take care of many people, approximately 1/3 of which are not eligible.

3—Indigent emergencies are already adequately taken care of in Baton Rouge Hospitals and there is adequate free state ambulance service daily and free state bus service to New Orleans Charity Hospital for non-emergencies.

4—The practice of medicine on the free enterprise basis is threatened as there are already about 3 free beds to each pay bed in Louisiana.

5—There is difficulty staffing the present hospitals and another one would only lead to greater shortage of nurses and ancillary personnel.

6—Baton Rouge is less than 100 miles from a charity hospital in any direction or about 2 hours by auto or ambulance. These hospitals are Independence, New Orleans, Alexandria, and Lafayette.

BOOK REVIEWS

Magnetic Removal of Foreign Bodies; by Murdock Equen, M. D., Springfield, Ill. Charles C Thomas, 1957, pp 94. Price $4.50.

Dr. Equen stresses an important procedure in bronchoesophagology. Since the days of Chevalier Jackson I have not seen such an extensive list of successful removal of foreign bodies from the esophagus and bronchi.

Dr. Equen is honest enough to admit his mistakes, and to include such adjuncts as biplane fluoroscope, antibiotics and tracheotomy. This method is much easier than the ones used prior to the invention of the Alnico magnet. I have seen Louis H. Clerf struggle with biplane fluoroscope and rigid gastroscope to remove with forceps a metallic foreign body from the stomach. Gabriel Tucker admitted spending $500 in designing a flexible pair of forceps for removing foreign bodies fluoroscopically from the stomach. He acquired a denture and a bobby pin. Credit should be given to Paul Holinger for the use of a magnet in esophagology and to Chevalier L. Jackson for use of a small magnet in the bronchioles.

The chest surgeon has encroached into bronchoesophagology almost transplanting the otolaryngologist. A general surgeon in a large clinic wailed that chest surgeons were trying to take over general surgery. Splenectomies and cholecystectomies were done through the chest wall. When a method for doing appendectomies through the ribs was devised, the general surgeons would be licked.

WILLIAM F. BONNER, M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, N. Y.: Rehabilitation in Industry, edited by Donald A. Covalt, M.D.

Louisiana State University Press, Baton Rouge: The Rudolph Matas History of Medicine in Louisiana, Volume I, edited by John Duffy.

Office of the Surgeon General, Dept. of the Army, Washington: Cold Injury, Ground Type, by Colonel Tom F. Whayne, MC, USA (Ret.), and Michael E. DeBakey, M.D.

W. B. Saunders Co., Phila.: Emergency Treatment and Management, by Thos. Flint, Jr., M.D. (2nd edit.); Callander's Surgical Anatomy, by Barry J. Anson, Ph.D., and Walter G. Maddock, M.D. (4th edit.).

Williams & Wilkins Co., Balt.: Human Parturition; Normal and Abnormal Labor, by Norman F. Miller, M.D., T. N. Evans, M.D., and R. L. Haas, M.D.